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FOURTEEN SIMULTANEOUS CASES OF AN ACUTE DEGENERATIVE STRIATAL DISEASE

NECROPSY IN ONE CASE REVEALING GROSS NECROSIS OF THE GLOBUS PALLIDUS (SYMMETRICAL) AND SUBSTANTIA NIGRA *

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One of the interesting but unsolved problems of brain pathology is the susceptibility of the striate body to damage from various noxious agents. That vascular accidents in the encephalon occur most often here is primarily a matter of hydrodynamics. Poelchen [1] sought to explain the damage to this structure following poisoning by carbon monoxid and other exogenous toxins as being due to the mechanical ischemia produced by proliferation of the intimal endothelium resulting from the circulating poison. He considered the selective effect on the striatum, especially on the globus pallidus, to be due to the greater precariousness of the blood supply to that part.

The tendency of the noxious agents in Parkinson's disease, Wilson's disease, pseudosclerosis and epidemic encephalitis to injure these centers selectively is now well known.

An outpatient in the Williams-Porter Hospital, Techow, North China, supposed to be suffering with epidemic encephalitis, informed the physicians that in his village and in his immediate family many other persons were sick in the same way as he was. We rode horseback twenty-five miles from the nearest railroad station and investigated the other patients and their surroundings. The cases illustrate the general susceptibility of the basal ganglia to injury, and raise interesting questions as to the nature of the process involved in these particular patients.

Following a winter of famine, 1920-1921, in northern Shantung Province, China, fourteen Chinese, children and adults, of that region

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* Read at the Fiftieth Annual Meeting of the American Neurological Association, Philadelphia, June 7, 1924.

1. Poelchen, R.: Cerebral Softening, Virchow's Arch. f. path. Anat. **112**:26, 1888.

were suddenly and nearly simultaneously attacked in February and March, 1921, by a malady the common symptoms of which were sudden giving way of the lower limbs, speech defects, convulsive seizures in some and disturbances of automatic associated movements in others. The adult patients recovered promptly; two children died; one has grown progressively worse during the intervening two years, and several are permanently crippled.

There were three families involved, all of whom maintained close social contact with one another. The Wang family, in which the first case developed, was not further invaded. The Liu family supplied nine cases, and the third family, surnamed Ma, four cases.

ENVIRONMENTAL CONDITIONS

For the three months before the outbreak, ordinary foodstuffs had failed, and all the villagers subsisted on a poor quality of maize, which they say was somewhat fermented. All of the patients, however, had been subject to only such famine and dietary conditions as thousands of their neighbors and fellow provincials, yet no other cases could be found in the neighborhood or province. Epidemic encephalitis was present in the province, but no cases occurred in the area now under consideration, unless some of these fourteen patients belong in that class. A peculiar form of fulminating myelo-encephalitis was present in various parts of North China that winter, though no cases were reported from this neighborhood.

REPORT OF CASES

Case 1.—Wang Hsiao Yun, a girl, aged 6 years, was drowsy, and for eight days slept continuously, although she could be aroused to take food, and at such times would talk intelligently. This lethargy then gradually decreased, but as the child became more alert, it was noted that she had difficulty in articulating. At the fourth week, her speech was unintelligible. Early in the third month, articulation began to be clearer. It improved during the following two years, so that when she was examined in March, 1923, she could be understood, although the dysarthria was still marked. During the second month of her illness, she found difficulty in coordinating lower limb movements. Slight spasms, which had first appeared the tenth day after the onset of the lethargy, were now distressing, and had spread to the trunk and upper limbs. The arms were rotated and the hands thrust up behind her back until they reached the scapulae; or at other times the hands rose to the chin. Her head was rotated on her neck, and her trunk twisted on its axis. These torsion spasms became progressively more severe and frequent until the child was exhausted by almost continuous convulsions during waking hours. They ceased during sleep. The second year, their force and frequency diminished.

When examined, March 30, 1923, the child was well nourished, alert and intelligent. She sat holding her left hand in the right, with the left elbow drawn far back. The digits were usually flexed, except the index and thumb, which were extended (Fig. 1). The eye movements and all cranial nerve movements were normal, the pupils equal and responsive. The left limbs

were feeble, the right showed good power. There was hypertonicity of the muscles of the left side, the tendon reflexes for that reason being elicited with difficulty. Ankle and quadriceps clonus and an extensor plantar reflex were present on the hemiplegic side. On the right side, muscle tone was somewhat increased; the tendon reflexes were above normal, and a less clear-cut Babinski toe response was observed. The neck muscles were rigid, and the patient's facial expression changed little during the examination. No tremor, vasomotor or sphincter defect was present, and there were no automatic laughing or crying movements. Her sensory condition was normal.

At times during the examination when she was passively moved, and always when she was picked up (Fig. 1 b), waves of muscle tone stiffened out her limbs, and her face was drawn tense. At such times, both upper limbs, especially the left, moved in slow, twisting spasms.

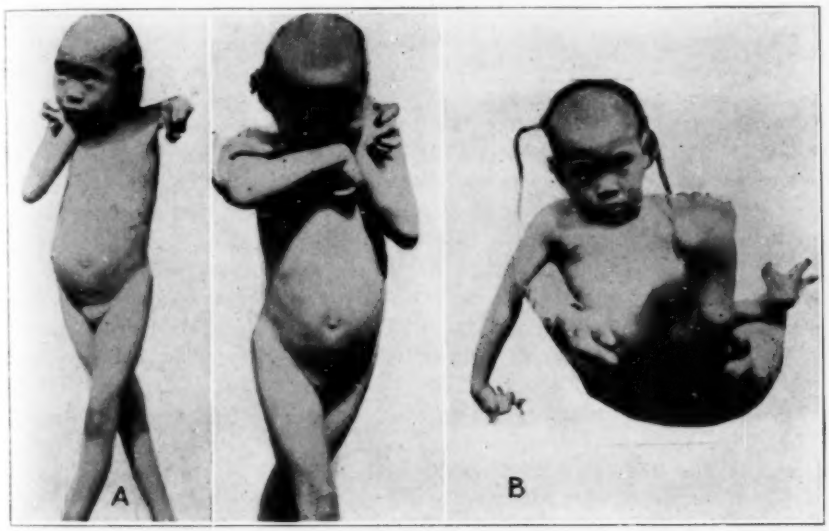

Fig. 1 (Case 1).—*A*, patient's effort to stand; various postures; *B*, becomes rigid in this position when held in arms.

The child was unable to walk or to stand without support. On any attempt to stand, her lower limbs were drawn in tight adduction, the feet assuming an equino-varus position.

Summary.—There was an acute onset two years before examination, with lethargy, dysarthria and torsion spasms. The patient became progressively worse the first year; then there was some improvement. Residual dysarthria, left hemiplegia, general hypertonicity of muscles, with convulsive seizures were present. The presumptive diagnosis was double corpus striatum involvement with implication of both internal capsules, especially the right.

Under ordinary circumstances, we should have considered this case as one of the forms of postinfectious encephalitis; but it must be considered together with the thirteen other cases that rapidly followed it.

Case 2.—A boy, aged 15 years, Liu Erh Yuan, lived near the Wang child, who frequently visited his home. The boy was in good health in early February, 1921, but, without preliminary symptoms, one day began to stagger

as he walked. He soon became sleepy and "talked queerly" (foolishly). There followed lethargy, dysarthria and, as the lethargy cleared, vertigo and some difficulty in vision. After twenty days he was out of bed, but for several weeks he walked "like a drunken man."

On examination, March, 1923, he was well nourished, intelligent and neurologically normal.

This group of symptoms occurs in otherwise identified cases of epidemic encephalitis.

Numerous brothers, cousins and other relatives of three generations lived with the Liu child in the same courtyard. Within a period of a few weeks, eight others of the family were affected with cerebral symptoms more or less like this.

Case 3.—An uncle of the patient in Case 2, aged 58, suddenly fell while walking. He rose and with care was able to reach home. He was weak, dizzy and unable to talk clearly for several days.

Case 4.—A younger brother of the patient in Case 3, father of the patient in Case 2, aged 36, while at work, felt his knees yielding under him. He fell, rose and tried to walk, but fell several times. He had vertigo, tinnitus and slight deafness; his "tongue felt thick," and his speech for several weeks was difficult to understand. He returned to work after twenty days. During that period and ever since he has been awakened from sleep once or several times each night by sudden muscular contractions, or "starts," a discomfort which he had never experienced before.

Case 5.—This patient, a son of the patient in Case 3, aged 22, staggered and fell as he walked home one day. He felt weak and sick, although neither he nor any of the preceding patients was aware of fever. He was in bed for six weeks. His complaints were of vertigo, "peculiar sounds in his ears" and inability to articulate clearly. He had completely recovered in March, 1923.

Cases 6 and 7.—A brother of the patient in Case 5, aged 17, and the seventh patient, an older brother of the patients in Cases 5 and 6, aged 34, were almost identically affected, but their illnesses were less prolonged.

Case 8.—The mother of the patients in Cases 5, 6, and 7, aged about 56, had a sudden attack of mental confusion, with dysarthria and insomnia. She was excited and required forcible restraint for several weeks. She was not completely well for two months.

Case 9.—A son of the patient in Case 4, a boy, aged 14 years, had an upper respiratory tract infection in late February, 1921, and felt ill enough to stay in bed two days. On rising, he could not stand without support. His speech became progressively slow and obstructed, and all his limb movements showed incoordination. The motor trouble increased until he was helpless and had to remain in bed or in a chair. Neither in his case nor in any of the preceding ones was there any pain or other sensory disturbance.

Examination.—Examination, March 30, 1923, revealed a boy in surprisingly good general condition, intelligent, and showing no sign of discomfort. His speech was slow, toilsomely delivered and difficult to understand. Saliva drooled from his partially open mouth. Uncontrolled movements of weeping and laughing occurred without appropriate emotional cause or on inadequate changes of feeling. He expressed interest in these queer performances.

When undisturbed from without, he lay quietly on his back, occasionally moving his muscles of expression. All observable movements of the involuntary

musculature proceeded without abnormality. His facial muscles at times assumed exaggerated grimacing aspects; at other times, an odd, quizzical expression produced by movements of the brows (Fig. 2 *A*).

There was moderate general hypertonia of the voluntary musculature, except of the forearms and hands, in which there were atrophy and flaccidity, and the calves, in which contracture had occurred with the production of pes equinus. On attempting to step, his feet were overflexed so that the big toes were knuckled under the foot. The tops of these toes were thus scraped on the floor, and thick callosities resulted. The hands were cold, moist and cyanosed like those of a patient with poliomyelitis.

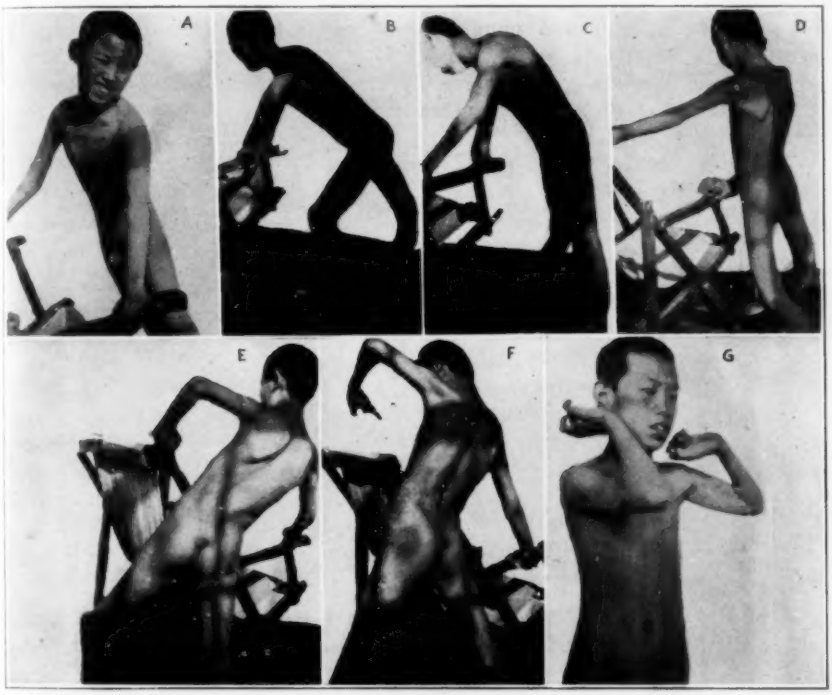

Fig. 2 (Case 9).—*A*, facial expression; *B*, *C* and *D*, hyperextension of knees; *A* to *F*, various attitudes assumed in progress around a chair; *G*, one phase of upper limb movements.

He ate with enjoyment, but invariably on receiving a mouthful, he pushed it into the left half of his mouth, the movements of his tongue becoming athetoid and rolling, so directed as to press the bolus against the left cheek. Yet voluntary protrusion and ordinary lateral movements of the tongue were well executed.

His facial musculature was in constant motion while he was conscious of being under observation, but otherwise these movements almost ceased. All the disordered movements stopped while he was asleep. Self-consciousness and excitement at once increased their severity.

When he was raised to his feet, his knees yielded in extreme extension (Fig. 2 *B, C, D*); his trunk rotated axially and bent laterally, and his upper limbs and trunk took on balancing movements like those of a man walking on a rail. He was able to walk by leaning heavily upon supports; but in the attempt, no orderly, automatic production of the elementary motions of stepping occurred. He effected each step by overcoming an apparent tendency to make inappropriate movements, toilsomely directing the limb by voluntary effort. He swayed in one direction until he seemed about to fall, whereupon he corrected the malposition by attention. Instead of moving his feet forward in courses nearly parallel with each other, he circumducted them in wide semicircles, then deliberately set them down. The movements reminded one of those of the awkward "stick insect" (plasma) (Fig. 2, *A* to *F*).

Neurologic examination revealed no paralysis of the cranial nerves or centers. The eyeball and pupillary movements were normal. There was atrophy of the forearm and intrinsic hand muscles and contracture of the calves, but no fibrillation. The pyramidal tracts showed no evidence of involvement. There was no tremor. He suffered no pain and showed no sensory abnormality.

There was no increase in cells or protein of the spinal fluid. The Wassermann reaction was negative.

Summary.—The onset of the disease occurred two years before examination with symptoms of a "bad cold." Then there was disruption of many of the highly organized motor syntheses, such as speech, chewing, walking and standing, and those of facial expression. In the case of certain intended voluntary movements, other and undesired movements were substituted or superimposed. There were no spasms or convulsions. Anterior horn degeneration was apparent in the cervical and lumbar levels.

The provisional diagnosis made in this case was of an acute degeneration of the lenticular nuclei and spinal anterior horn cells. The etiology was unknown; possibly the condition was a postinfectious encephalitis. (This patient was examined a year later — March, 1924 — and was found to be weaker, but otherwise in about the same condition as above described.)

Case 10 will be described below with the necropsy findings of the brain and spinal cord.

Case 11.—Ma Chiao Erh, aged 8 years at the onset, March, 1921, was one of four patients in his household, the only one available for examination in March, 1923. He became sick at the same time as the Liu and Wang patients, and was thought to have shown slight fever, although thermometers were unknown to these villagers. The first clear symptom was "thickness of speech," and then paralysis of all four limbs. He is said to have lain in bed two weeks in a lethargic state, in which "he moved his eyes about as people came and went." After a month, he was able to talk clearly and to walk. He did not have spasms or convulsions; but when passively moved, his lower limbs became rigid.

When examined, in 1923, this boy was in good condition and mentally alert. His lower limbs soon yielded under him when he tried to stand. There was slight hypertonicity of the trunk and limb musculature, and waves of increased tonicity swept over his lower limbs when disturbed by the examiner or when he tried to stand. The limbs gave slightly increased tendon reflexes with clonus of the left ankle, and extensor plantar reflexes on both sides. The pyramidal tract defect was more marked on the left side. Contracture of the calves and pes equinus were present. There was a left internal strabismus,

but unfortunately the parents could not remember whether it had existed before the onset of the present disease or not. The pupillary and all other cranial nerve movements, except those of the left external rectus, were normal. There was no sensory, sphincter or trophic anomaly except the contracture in the calf muscles.

We should ordinarily have set this case down as one of the encephalitic or vascular accidents of childhood, with crippling of the internal capsules. But three other members of the patient's family were involved in succession: his grandfather, aged 53; an uncle, aged 20; and a sister, aged 9. Each was suddenly seized with "weakness of the knees" and inability to enunciate clearly; and the survivors showed peculiarity of gait after recovery. The sister at the onset was unable to utter words. She had spasms of the limbs in which she would twist her arms, carrying her hands up her back, and rotate her trunk to one side or the other. The convulsions became progressively more severe until her death within four weeks after the onset.

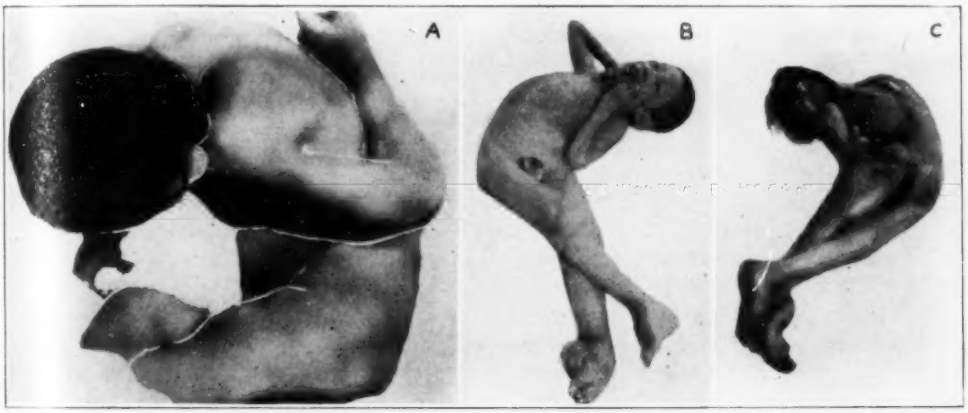

Fig. 3 (Case 10).—*A*, spasm in sitting position; left upper limb drawn to scapula, legs crossed; *B*, lying on bed, one hand approaching mouth; *C*, convulsions at times turned him face downward.

From the description given by her parents and the neighbors, this girl's condition greatly resembled that of the tenth patient, now about to be described, except that the course was more acute and death more prompt.

Case 10.—*History.*—This patient, aged 5 in 1921, was the younger brother of the patient in Case 8. While playing, he fell because "his knees gave way." He rose and stood for a moment, then suddenly fell again. This continued until he was put to bed merely to protect him from falling, not because of any other discomfort. Ten days later, spontaneous spasmodic movements set in, with the same peculiar twisting of the upper limbs observed in the other patients, in which his hands were carried to his scapulae (Fig. 3 *A*). The spasms continued, spread to the axial trunk muscles and to the lower limbs. They became progressively more severe. On the fifteenth day, difficulty in speech was noted. No eye symptoms, sphincter trouble, lethargy or sensory trouble occurred.

By the twenty-second month, the torsion spasms had become so severe that he could not sit up. His parents would press on his back, raising him into

a sitting posture because of real or imagined relief the position afforded him during the height of the convulsions. During one such performance, his right femur was fractured. It healed, but shortly before our examination, the left femur was similarly fractured, and it had not united at his death. The convulsions became more and more severe and continuous, until the quiet interval had been reduced to only fifteen minutes during waking hours. At night, he slept fitfully, and would be awakened by new convulsions after short periods of rest.

Examination.—Examination, March 30, 1923 (three days before death), revealed an intelligent, quiet, responsive child of about 7 years of age. He lay in bed and had an anxious expression, which became agonized at the approach of new spasms. These attacks recurred every fifteen minutes and continued from two to ten minutes, and then relaxed only partially. During the convulsions, his lower limbs were crossed in strong adduction, his trunk was bent sharply to the left (Fig. 3 *B*), the thorax was drawn strongly upward, and the diaphragm and abdominal muscles held as if he were straining at stool. His neck was not rigid, but relaxed. It was rotated so that the chin approached the left shoulder. The paroxysms resembled those of tetanus, except that the neck and jaw muscles were less affected.

Course of Illness.—A peculiar feature of the disease was that the hands were at times not drawn up to the scapulae, but to the clavicles; and at such times were then pressed into the mouth. He bit through the soft tissues, and chewed his hands until they were deeply denuded. Gauze bound over his hands to prevent these wounds became soaked with saliva, adding the marks of maceration and fostered pus infection. The nails were loose, and one had sloughed off. This biting of his own tissues appeared to give him some sort of satisfaction during the convulsive exacerbations.

The child evidently suffered severely during the spasms, but made little outcry. The pain from the fractured femur and the wounded hands was submerged in the severer pain of the muscular contractions.

Permanent deformities had resulted: his chest was barrel shaped, with the sternum to the left of the sagittal plane. The ankles were held in strong flexion with pes equinus (Fig. 3 *B*). The calf muscles were contracted. The general muscle tone was high, but there was no evidence of pyramidal tract involvement. His cranial nerves and lower motor centers functioned normally. All the muscles were wasted, but without fibrillation. The sphincters were well controlled up to a few days before death. Between paroxysms, he talked intelligently, although he seemed taciturn. He uttered his words rapidly in gusts interspersed with gasps, and his speech was difficult to understand. Rectal temperature rose during the last days to 42 C.

Wassermann tests of the blood and spinal fluid were negative. Red cells were present in the fluid so that no significance was attached to the slightly raised white cell count. Cultures from the spinal fluid were negative.

Respiration became irregular, and the patient died, March 31.

Necropsy.—Necropsy was allowed by the family on the nervous organs, but not on those of the abdomen or thorax. Formaldehyd was injected through the carotids immediately after death pending permission for further examination. Immediately on opening the neck, a peculiarly disgusting odor came from the tissues.

The pia-arachnoid was edematous, the fluid brownish. The epidural fat was like a dark jelly. The leptomeninges were slightly thickened and somewhat grayish. The ependymal cavities throughout were moderately

dilated. Yellow spots of necrosis were found in each globus pallidus (Fig. 4) and in the left substantia nigra, just between that structure and the pyramidal bundle (Fig. 5). Spinal cord sections revealed no gross changes.

Histopathologic Examination of the Brain and Spinal Cord.—The carotids and their cerebral branches showed no thickening or infiltrations. The

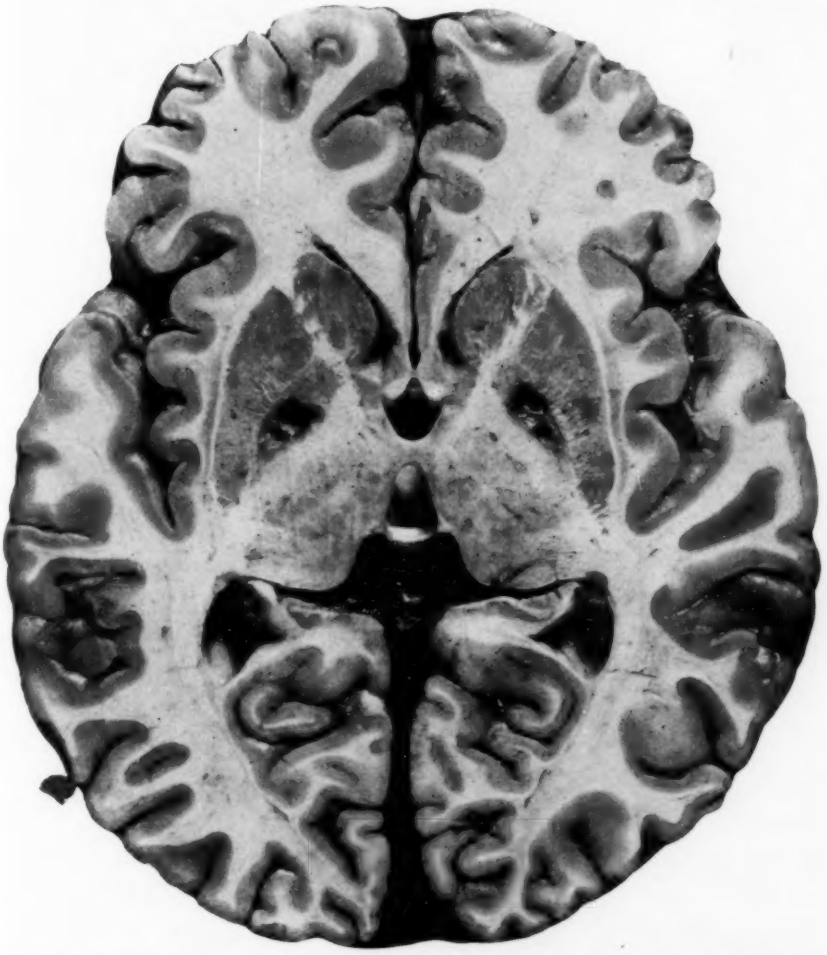

Fig. 4 (Case 10).—Section at level of massa intermedia of the thalami, showing necrotic spot in each globus pallidus.

smaller blood vessels of the brain and spinal cord were normal, except for a moderate dilatation of the lumen and thinning of the walls. (Formaldehyd had been injected postmortem). There was no sign of intramural or perimural infiltration. The stroma around many smaller blood vessels was rarefied, especially in the striatum. A few small, agonal hemorrhages were present. Around the vessels of the pia at all points were extravasated red cells. There were no gross hemorrhages. There was no thrombosis.

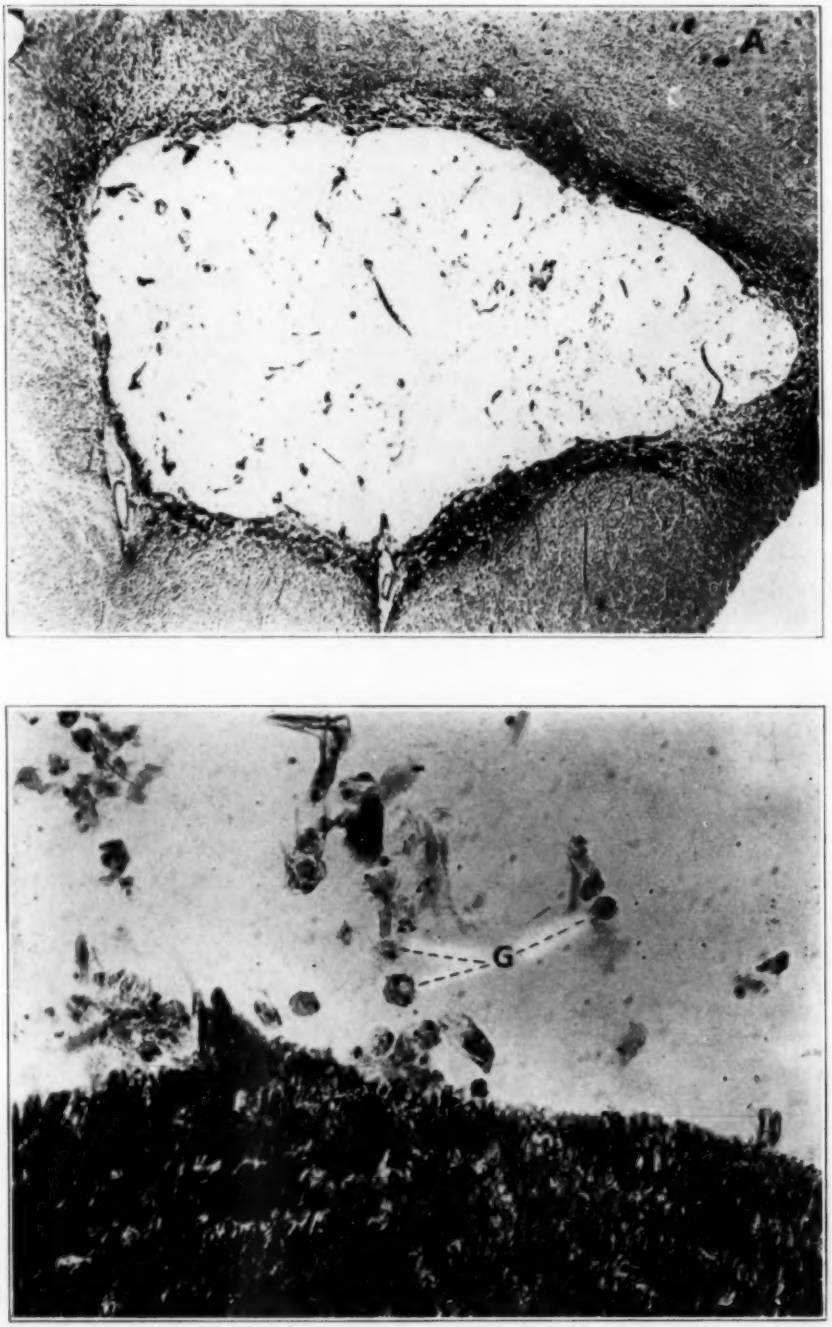

Fig. 5.—*A*, necrotic spot in substantia nigra, showing capsule-like glial condensation. (Phosphotungstic hematoxylin stain; × 50.) Lower illustration shows edge of same; surprising evenness, almost as if fibrils had been cut by scissors. Gitter cells at *G.*; × 750.

Throughout all levels, *the membranes* showed a mild inflammation, with mononuclear cells and connective tissue elements, and with slight thickening. But at the upper medulla oblongata there was an intense inflammatory process, most marked in and near the ventral median sulcus. The pia-arachnoid was here very thick and densely infiltrated with mononuclear and polymorphonuclear cells. The walls of the blood vessels shared in this infiltration, and their intimal coats showed dense endothelial proliferation, the new cells pressing out and in some sections almost filling the lumen. Around the vessels were many points of cell necrosis like that seen in tuberculous meningitis. Various special stains, however, showed no organisms (Fig. 6).

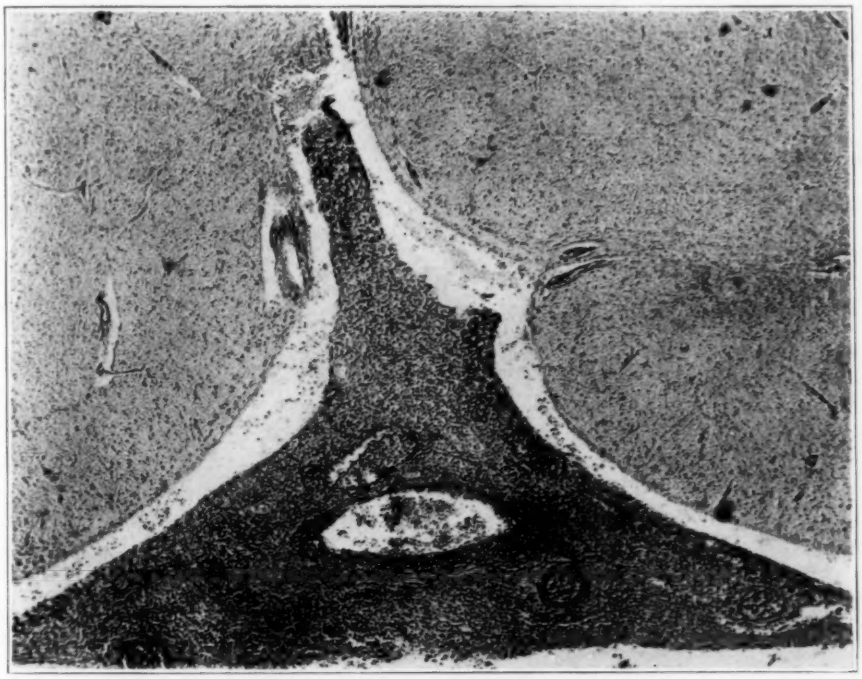

Fig. 6.—Dense infiltration of pia-arachnoid in the ventral sulcus of the oblongata. Eosin-hematoxylin stain; × 80.

The areas of gross softening occupied, in vertical extent, the lower half of each globus pallidus; in dorsoventral extent, about the posterior two thirds of those structures. The globus pallidus in this patient was not subdivided by a medial lamina. The necrotic spot in the left substantia nigra was 3 mm. in transverse diameter by 2 mm. dorsoventrally (Fig. 5).

These cavities were like those of ordinary thrombotic softening: irregular walls with trabeculae of partially destroyed tissue at their borders. Blood vessels passed through at the margins, some of which were almost denuded of glial support. In the cavities and in the surrounding tissues were myriads of gitter cells loaded with fat droplets. Around the cavities were dense glia walls resembling capsules. Only in these areas was new blood vessel formation observed. The process here appeared to be reparative.

The neuroglia showed some abnormality. The cytoplasm of the cells was increased and more deeply stained than in normal cells. There were unusually large numbers of small, oval, very pale nuclei (Fig. 7) containing nucleoli; also many larger nuclei, varying in size from two to four times the diameter of resting glia nuclei. Dense aggregations of nuclei occurred as nests (Fig. 8) in the lower levels of the striatum, some of these aggregations being composed of large nuclei containing much chromatin, others of the very pale, oval variety. There were no excessively large, "giant," glia elements, and the glial cytoplasm contained no pigment inclusions.

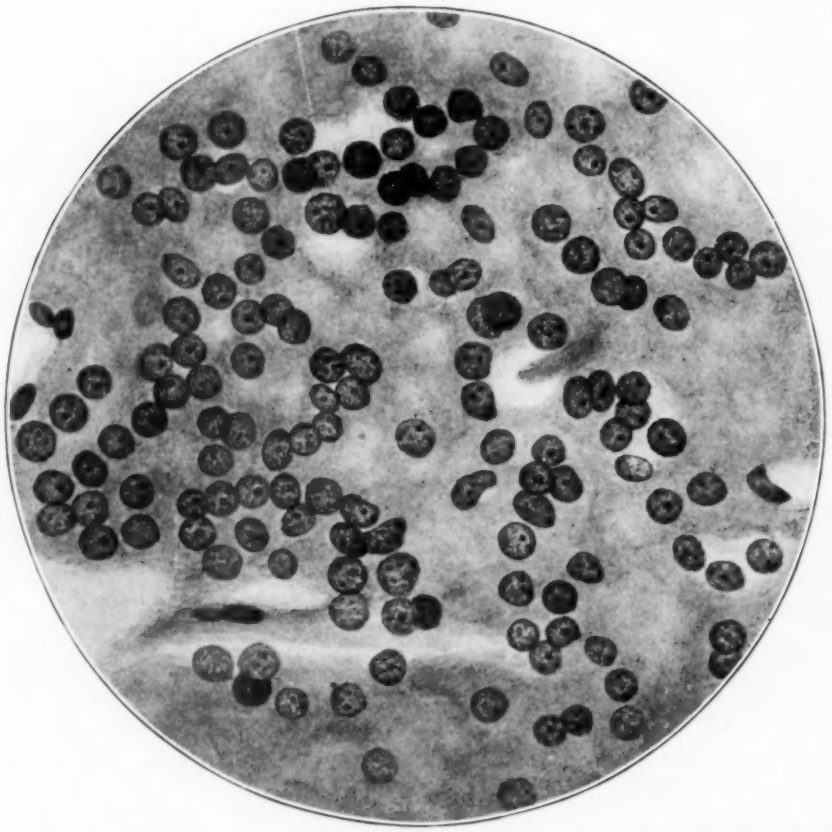

Fig. 8.—Nest of glial cells in striatum; the nuclei are deeply stained with hematoxylin. (Drawn from oil immersion field.)

Necrosis of the ganglion cells was present in all parts examined, but it was most pronounced in the substantia nigra and striate body. Here it was intense; large and small cells were vacuolated, their cytoplasm being ragged at the border. At times half or more of a cell was completely gone (Fig. 9.) Lipoid stains showed fat droplets of various sizes within and around these cells. Often large or small ganglion cells were represented by only a spongy mass of débris.

Glia cells were found within the broken-down cytoplasm and packed in the vacuoles of these cells, or in the irregular reentrant spaces of their borders.

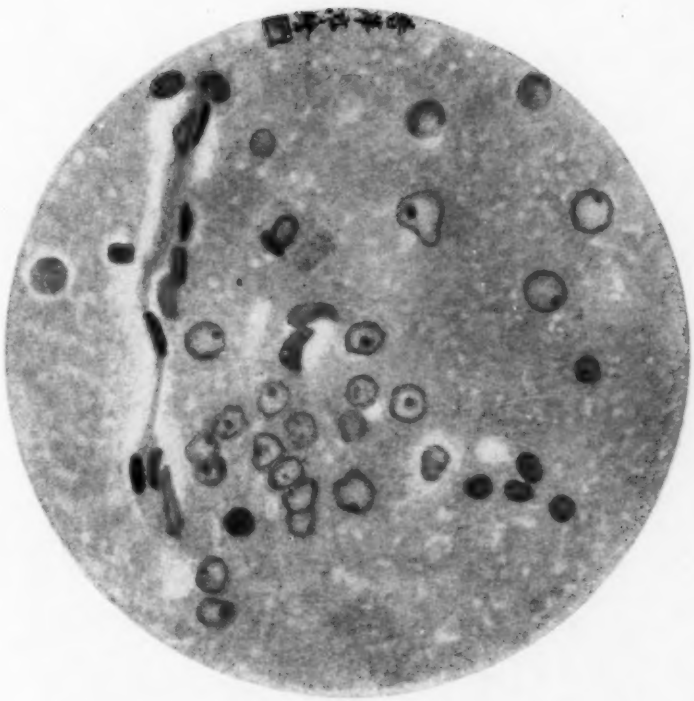

Fig. 7.—Pale glia nuclei from caudate nucleus; diameter twice to three times that of ordinary glia nuclei, of which several specimens are present. These pale nuclei often resembled those of ganglion cells. (Painted from oil immersion field. Hematoxylin-eosin stain.)

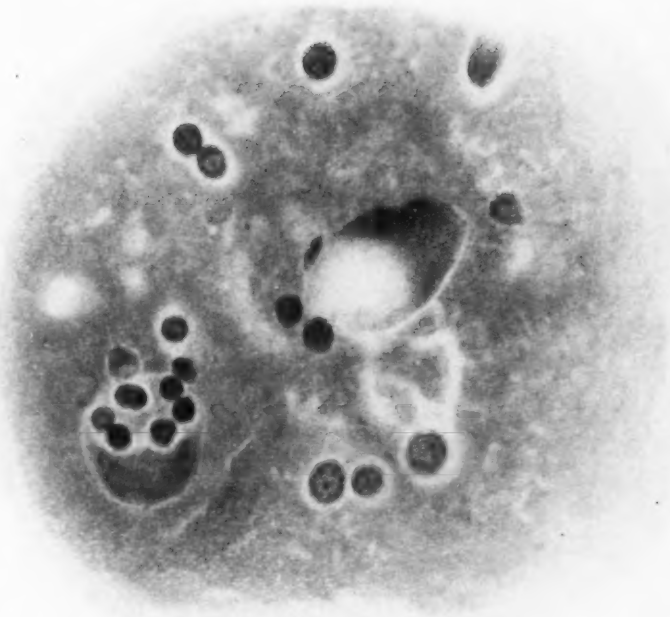

Fig. 9.—Necrosis of ganglion cells in striatum (Painted from oil immersion field. Hematoxylin-eosin stain.)

The ganglion cell death was less marked in the brain stem and spinal cord, but every field showed some seriously affected cells, as shown by altered outlines, fragmented cytoplasm, displaced and altered nuclei and poor staining capacity of cells; also in sharlach preparations, in all of which lipoid appeared in the nerve cells in clumps or droplets. According to Spielmeyer,[2] the nerve cells of a child of 7 years should show no lipoid pigment.

The ependyma from one end of the nervous system to the other, especially that nearest the zones of maximum cell death, had fat droplets of various sizes under and within its cells, and lying on its free surface. The apparent movement of fat toward both blood vessels and the ependyma, as if the fat were being conducted from the nervous tissues toward blood and lymph channels, has been pointed out by Hassin.[3]

Summary of Pathology.—The meninges showed a slight, generalized leptomeningitis which was intense in a limited area at the upper level of the medulla oblongata. There were edema of the membranes and many red blood cells within the membranes.

There was hydrocephalus of slight degree.

The blood vessels showed capillary sprouting only near the areas of softening. There were no hemorrhages or thromboses and no perivascular infiltrations. The stroma was rarefied near the small vessels.

There was fibrillar glia proliferation around areas of softening and there was a general increase of glia cells. The cell bodies were moderately enlarged, many with large, pale-stained nuclei. There were peculiar nests of glia cells in the striate bodies but no giant glia cells.

The ganglion cells showed widespread necrosis, most intense in the substantia nigra and the lower levels of the striate body. The cytoplasm was vacuolated and often contained neurophages, and there was lipoid pigment in and around degenerated cells.

There was no tract degeneration.

There was gross softening in the globus pallidus of each side and in the left substantia nigra.

CLINICAL SUMMARY OF THE FOURTEEN CASES

1. The onset was abrupt, without fever in thirteen cases. The first symptoms were repeated failures of the muscles of equilibration and then speech defects without aphasia.

2. Three of the patients had lethargy lasting from a few days to more than a week. Seven complained of vertigo and faulty hearing.

3. All showed disorders of the highly synthetized movements, some merely irregular waves of muscular tone, some torsion spasms, and some had athetoid and balancing movements superimposed on attempted voluntary movements.

4. There was concomitant internal capsular involvement in two patients.

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2. Spielmeyer, W.: Histopathol. des Nervensystems, Berlin, 1922, p. 28.

3. Hassin, George B., and Bassoe, P.: Myelitis and Myelomalacia, Arch. Neurol. & Psychiat. **6**:32 (July) 1921, and elsewhere.

5. The course was abortive in five, of brief duration in four, with practically complete recovery in all of these; it was progressive without death in three, with death after two years in one. One case was fulminating, with death in two weeks.

6. The age range was from 4 to 56 years. The patients with fatal cases were the youngest; no patient over 20 years of age was seriously ill. Both the severity of the symptoms and the fatality were in inverse proportion to age.

7. Three of the cases occurred in females, of whom one died; eleven occurred in males, of whom one died.

NATURE OF MOTOR DISTURBANCE

The preliminary motor symptom was a sudden, recurring loss of normal postural or antigravity muscle tone. In eight of the fourteen patients, recovery of motor control was complete by the time we examined them. In one other, the only residual motor trouble was a tendency to "start" during sleep, apparently a persistence of the disturbance of the tone mechanism. In two patients, pyramidal tract involvement complicated the study of their motor condition; but both of them at times still showed torsion movements. In two others, the torsion spasms persisted and progressed in severity until death, and were so violent and constant that any study of the degree of motor coordination between the spontaneous convulsions was impossible. In one (Case 9), when voluntary movements were attempted, in place of the well ordered movement desired, other well enough coordinated movements resulted, but not the ones intended. These were of a waving, rather graceful nature, somewhat like those of a danseuse. It looked as if the combination desired could not be effected, but another, nearly related combination was put into operation instead.

Taking the cases all together, there was an early variation in the postural tone control, causing sudden loss of tone at times, and sudden accessions of tone at other times. There was, in addition, some degree of pure loss of striatal motor synthesis, but this was difficult to evaluate, being masked by the frequent recurrence of positive, coordinated but inappropriate movements. These undesired movements were mild in some patients; in others, convulsive (torsion spasms).

The internal capsular and anterior horn involvement were coincidental and of secondary importance.

The absence of certain symptoms was significant: There were no intellectual faults, sensory troubles, myoclonus, tremor, fibrillation, cranial nerve paralysis, parkinsonian symptoms, evidences of liver trouble or corneal changes.

COMMENT

While the areas of gross softening in the one case that came to necropsy resembled those found after cerebral hemorrhage or thrombosis, there was found no such vascular basis, and the symptoms and manner of onset in all the cases are out of harmony with such an explanation.

A. Encephalitis.—No sign of inflammatory infiltrations was found around blood vessels or elsewhere, except in the meninges. In a remarkable family group reported by Paterson and Carmichael,[4] small cell infiltration of the lenticulae, with glial proliferation and absence of nerve cells, was attributed to the spread of an intense septic process in the membranes around the peduncles. But in our case, no such spread of the inflammatory process was present. The meningitis seemed to be a coincidence, possibly a terminal infection.

Subsidence of the perivascular and diffuse cell collections of epidemic encephalitis was demonstrated in cases studied by Spatz,[5] but his cases were chronic and had come to necropsy two or more years after the acute period. Our patient, after a mild onset had shown signs of an increasingly active process, which progressed to death and yet showed no inflammatory infiltration.

Gross necrosis has not to our knowledge been demonstrated in clear cases of epidemic encephalitis.

That fourteen persons were attacked simultaneously in one small community, argues against epidemic encephalitis. Neighborhood hospital or school epidemics have been reported by MacNalty[6] and others, but in such instances the cases were typical and the infection appeared to have been passed successively from earlier patients to others. Stiefler's[7] study of a large number of cases published in the general literature confirms the belief held by most observers from the beginning that epidemic encephalitis is rarely transmitted from an infected person to a group in his immediate neighborhood. Also in our cases *the absence of acute febrile onset, of root pains,* cranial nerve paralyses, myoclonus, and parkinsonian sequelae, taken with the fact that no typical cases of epidemic encephalitis were known in the villages near at hand is noteworthy.

We conclude, therefore, that our cases are not to be classified as encephalitic, either of the epidemic (lethargic), or the postinfection type.

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4. Paterson, D., and Carmichael, E. A.: Brain **47**:207, 1924.

5. Spatz, B.: Report of Transactions of the Danzig Neurological Conference, 1923.

6. MacNalty, A. S.: Public Health and Medical Subjects, Reports of British Ministry of Health, No. 11, 1922, p. 44.

7. Stiefler, George: Zur Frage der Kontagiosität der Encephalitis epidemica, Ztschr. f. d. ges. Neurol. & Psychiat. **42**:396, 1922.

B. Pseudosclerosis and Wilson's Disease.—For clinical reasons and reasons of pathology, the cases under consideration are not to be classed under this name. They differ from classical cases of the pseudosclerosis group in the abruptness of onset, the fulminating course in one patient, the number of persons simultaneously involved in a small area, the occurrence of abortive cases, and in the absence of tremor, hepatic symptoms, corneal changes and psychic deterioration. The gross necrosis involved the globus pallidus and substantia nigra, not the putamina. The essential histologic change, viz., the giant glial cells of the Alzheimer type, was not present.

Yet these Chinese cases possess certain significant features in common with cases otherwise recognized as Wilson's disease or pseudosclerosis. The process was noninflammatory and degenerative, leading to severe necrosis of ganglion cells together with reactive glial changes. These elements were more severely injured in the striate body and its downward extension, the substantia nigra, but they suffered also in all parts from the cortex to the lumbar spinal cord. The symptoms, like those of pseudosclerosis, consisted in derangement of the myostatic mechanism and of the automatic associated movements, the derangements being both positive and negative. The severity of the process varied inversely as the age of the patients.

The resemblance is striking, for instance, between our Cases 9 and 10 and a case reported by von Economo.[8] This boy was 15 years old, without familial history of nervous disease. While in good health, he fell because his legs gave way. Fragility of bones, anarthria, fixity of facial expression, drooling, derangement of muscle tone, signs and symptoms of mild meningitis and atrophy of calf muscles with pes equinus marked the course of his illness. Necropsy showed a noninflammatory degeneration of the lenticular nuclei, dilatation of ventricles and glial proliferation. There were enlarged glia cells, but none of giant size. The ganglion cells were broken down. This patient's condition, however, conformed to the classical description of Wilson's disease because of the occurrence of hepatic cirrhosis and psychic deterioration, and because the softening was in the putamina (not, as in our case, in the pallida). The patient also had pulmonary tuberculosis.

Some of the minor pathologic features of our Case 10 have also been found in cases of pseudosclerosis. Dilatation of the ventricles was found by Hösslin and Alzheimer[9] and by von Economo.[8] Wilson's

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Psychiat. **8**:183, 1912.

8. Von Economo Wilson's Disease and the Syndrome of the Corpus Striatum, Ztschr. f. d. ges. Neurol. u. Psychiat. **43**:173, 1918.

9. Hösslin, C. V., and Alzheimer, A.: Ein Beitrag zur klinik und patholog. Anat. der Westphal-Strümpellschen Pseudosclerosis, Ztschr. f. d. g. Neurol. u.

original cases showed in several instances "patchy meningitis." Fragility of bones, atrophy of leg muscles and pes equinus were observed by von Economo in the case above cited.

But the pathologic criterion which, according to Spielmeyer [10], distinguishes both Wilson's disease and pseudosclerosis from other disease entities, is the combination of ganglion cell necrosis with the peculiar glia changes described by Alzheimer. These glia cells probably show phasic variations, as pointed out by Westphal and Sioli,[11], the huge elements not being present at all stages. But the presence at least of large nuclei, often multiple, marked by lobulations and foldings of the nuclear membrane, and surrounded by cytoplasm containing pigment inclusions, must be demonstrated before this diagnosis is warranted.

The absence of these typical glia cells in our case, in which necropsy was performed, rather than minor differences in the clinical manifestations, is the reason for not including it under this classification. For, as has been pointed out by others, wide variations in motor symptoms are to be expected in a group of diseases characterized by irregular cell destruction within the striate body and its functionally associated centers, including the substantia nigra and dentate nucleus. Cases may present even such varied clinical pictures as those of Sawyer,[12] Thomalla [13] and Wimmer,[14] yet since the fundamental histologic changes of pseudosclerosis were present, the diagnosis was established.

C. Toxic Degenerations.—Exogenous poisons, such as carbon monoxid, manganese, lead and spoiled protein food could not be discovered as etiologic agents. The liability of the basal ganglia to injury from these substances was kept in mind during our examination, but the mode of life of the villagers and the character of the pathologic findings give no basis for suspecting this class of poisons.

Endogenous Poisons.—Our conclusion is that the pathologic condition in these patients was a toxic degeneration. A sudden noninflammatory, progressive process, causing necrosis of ganglion cells, reactive glial changes, dilatation of blood vessels with rarefied zones around the vessels, and without demonstrable bacteria in the tissues or spinal fluid, suggests the action of a circulating poison.

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10. Spielmeyer, W.: Die Histopathology, Zusammengehörigheit, etc., Ztschr. f. d. g. Neurol. u. Psychiat. **57**: 312, 1920.

11. Westphal, A., and Sioli, F.: Clinical and Anatomical Contribution to the Subject of Pseudosclerosis, etc., Arch. f. Psychiat. & Neurol. **66**:747, 1922.

12. Sawyer, J. E. H.: A Case of Progressive Lenticular Degeneration, Brain **35**:222, 1913.

13. Thomalla: Torsion Spasm, Ztschr. f. d. ges. Neurol. & Psychiat. **41**: 311, 1918.

14. Wimmer, A.: Extrapyramidal Symptoms, Rev. neurol. **28**:952-1206, 1921.

Wilson, and many later writers, in discussing the possible origin of Wilson's disease and pseudosclerosis, felt that some as yet undiscovered toxin was the causal agent. In von Economo's case and in an instructive case reported by Spielmeyer, active tuberculosis was present, and aroused the suspicion that its toxin might be causally related to the other changes. Westphal and Sioli reported a case with exhaustive anatomic studies, in which the symptoms of "grip" had been followed first by those of encephalitis and later by those of pseudosclerosis. Necropsy showed some of the changes of epidemic encephalitis in a picture otherwise that of pseudosclerosis. The writers cautiously suggest that a toxic-infectious process such as "grip" might be responsible for the production in the body of toxins that could in the earlier stages cause the lesions and symptoms of epidemic encephalitis, and later those of pseudosclerosis. We are unable to follow their reasoning to such a final conclusion, but are ready to believe that an original infectious process might lead to malfunctioning of organs and the production of secondary toxins, which in their turn would cause such changes as we have described. It would, however, be mere speculation were we to attempt to associate the famine conditions, the influenza-like attacks and other respiratory tract infections, to which the Chinese are constantly subject as etiologic factors in our cases.

SUMMARY AND CONCLUSIONS

1. Without traceable familial, infectious or other disease, or the action of external poison, fourteen persons in three families were simultaneously affected with a disease of rapid onset, exhibiting disorders of striatal motor functions.

2. In the one case that came to necropsy were found death of ganglion cells, glial reaction and gross necrosis, the process being most severe in the substantia nigra and globus pallidus. There were no evidences of encephalitis.

3. It is established that certain toxic agents effect preponderant damage on the striatum and substantia nigra while injuring other parts of the nervous organs to a lesser extent. Among these are: Certain exogenous poisons, e. g., carbon monoxid; the virus of epidemic encephalitis, probably also the viruses of some other infectious diseases; unknown poisons, probably of endogenous origin: (*a*) of these, some affect, in the nervous system, the tissues both of mesodermal and ectodermal origin; (*b*) others affect solely, or preponderantly, the tissues of ectodermal origin, i. e., neuroglia and ganglion cells.

4. Among the toxic diseases causing selective damage to the nervous tissues of ectodermal origin, Wilson's disease and pseudosclerosis form a distinct pathologic entity. But it seems probable that a number of

other diseases marked by striatal symptoms and reported as forms of epidemic encephalitis, really belong in this class of noninflammatory, toxic diseases, and are thus closely related to the pseudosclerosis group. The cases reported by us appear to belong in this class, and are a hitherto unknown subvariety of it.

DISCUSSION

Dr. J. Ramsay Hunt, New York: I agree with Dr. Woods' interpretation to a great extent. It seems to me that the case belongs rather in the pseudosclerosis group. Some of the glia cells which he showed in the last picture were rather swollen, hydropic cells, suggesting a form of the Alzheimer cell.

Are some of the glia cells of the Alzheimer type? Were there changes in the cerebellum, in the region of the dentate nuclei?

Of course, in a case like this, while the voluntary movements are most interesting, I do not believe that we can draw any important conclusions, as so many structures are involved. I think in the elucidation of the problem of involuntary movements in the region of the midbrain and the substriatal region, we will have to depend on limited vascular lesions, or the system types of disease.

Dr. A. H. Woods: I am glad Dr. Hunt called attention to the fact that I did not make the matter of the distribution of the lesions sufficiently clear. The pathologic changes referred to were widespread throughout the nervous system, from the lumbar spinal cord upward. They consisted in degeneration of ganglion cells and alterations as to form, size and number of the glia elements. Some glia cells were very pale and two or three times the ordinary diameter of such cells. But there were no "giant" glia cells.

As to localization of function, the involvement was so general that no definite inferences as to function should be attempted from such cases. In speaking of the motor disturbances as "striatal," I depended on findings derived from published cases which presented more definitely limited lesions.

Dr. Hugo Mella, Boston: Were these patients exposed to any metallic poisoning, such as lead?

I have recently seen two cases showing a typical postencephalitic parkinsonian syndrome, in both of which a large amount of lead was found in the stools—amounts large enough to justify making a diagnosis of lead poisoning.

One patient was a young man who gave a typical history of acute encephalitis of the epidemic type, but in going into his history a little more carefully, I found that he had been dipping golf balls for some years in white lead, working it in with his hands. He improved from this acute attack, after being in bed for about six weeks. About five months later, the parkinsonian syndrome developed. The question came up as to whether the acute attack may have been a lead encephalopathy rather than an epidemic encephalitis, and that there are now vascular changes in the basal ganglions producing the parkinsonian syndrome.

The second case, that of a woman, aged about 43, gave practically the same history as to the onset of the parkinsonian syndrome. A large amount of lead was found in the stool. In looking for a source, I could find nothing in the home—it was a modern house, so far as plumbing was concerned. I found, though, that she had been using a certain face powder for some years. An examination of the powder revealed a large amount of lead.

Dr. Andrew H. Woods: There were no giant glia cells. The ganglion cells of the dentate nucleus were moderately affected.

I examined the tissues postmortem, and had previously examined the domestic arrangements and dietary habits of the patients for the possibility of carbon monoxid, alcohol, arsenic and lead poisoning. The vessels they use in cooking were earthenware and unglazed. Their food was largely farinaceous. There were no water pipes or other detectable sources of mineral poisons.

As I said in speaking of the pathologic findings, there was no sign of inflammation in the tissues. The patients had exhibited no eyeground changes, and the early symptoms as described did not suggest neuritis or gastro-enteritis. Our own examinations showed the absence of such changes later.

DECEREBRATE RIGIDITY IN MAN*

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Our knowledge of muscle tone has been enriched by the study of experimental decerebrate rigidity. Recently many clinical cases have been described in which the condition of the patient was said to have fulfilled completely or only in part the requisite characteristics of that condition.

The anatomic and physiologic considerations involved in decerebrate rigidity have been emphasized recently by Walshe [1] and by Pollock and Davis.[2] It is clear that if we are to apply the condition of decerebrate rigidity to clinical cases, certain anatomic and physiologic requirements must be met. Anatomically, of course, the complete experimental lesion will never be attained in the clinic. On the other hand, the essentials of that lesion may be obtained; namely, interference with voluntary motor power and abolition of postural reflexes in the midbrain. Physiologically, there should be a true hypertonus which selects the extensor muscles of the extremities, and this should be accompanied by reciprocal flaccidity of the flexors. This rigidity should have the qualities of experimental decerebrate rigidity; that is to say, the rigidity should be plastic. This characteristic may be brought out by the demonstration of sudden yielding of the rigidity on passive stretching of the muscle or by the prolonged tendon reflexes which may be elicited. Further, it should be possible to offset the rigidity by phasic spinal reflexes. The exhibition of the tonic reflexes described by Magnus and deKleijn are not necessary to the presence of decerebrate rigidity. Their presence in the absence of postural reflexes, however, adds strong evidence to the existence of such a condition.

Magnus and deKleijn [3] have briefly reported several cases of clinical interest. They were, of course, concerned primarily with the presence of postural and tonic reflexes and not with decerebrate rigidity.

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* The material for this article was obtained in the Surgical Clinic of Dr. Harvey Cushing, Peter Bent Brigham Hospital, Boston.

1. Walshe, F. M. R.: Decerebrate Rigidity in Animals and Its Recognition in Man, Proc. Roy. Soc. Med. Sec. Neurol. **15**:41, 1922.

2. Pollock, L. J., and Davis, Loyal E.: Studies in Decerebration. II. An Acute Decerebrate Preparation, Arch. Neurol. & Psychiat. **12**:288 (Sept.) 1924.

3. Magnus, R., and deKleijn, A.: Weitere Beobachtungun über Hals und Labyrinthreflexe auf die Gliedermuskeln des Menschen, Pflüger's Arch. **160**: 429, 1915.

The greater number of their cases are so incompletely reported that it is extremely doubtful that they can be considered as clinical examples of decerebrate rigidity. In 1920, Wilson [4] reported a series of cases which he divided rather generally into those of decerebrate rigidity with and without tonic fits. He also expressed the opinion that many of the involuntary postures of a limb in various nervous diseases, as well as the transient positions assumed in chorea and athetosis, are parts of the decerebrate attitude. If one carefully considers each case reported by Wilson in the light of the experimental decerebrate animal, one must of necessity rule out many of his case reports as examples of true decerebrate rigidity. Tonic fits do not occur in the decerebrate animal except as a result of hemorrhage or undue irritative trauma to the medulla. The decerebrate animal has a gradual loss of thermic control without any irregularities of pulse or respiration. Further, the assumption of an attitude similar in some respects to the decerebrate rigidity of experimental animals is not alone sufficient to classify that case as an example of partial or complete decerebration. Strychnin poisoning and meningitis produce an attitude similar in appearance to that of the decerebrate animal, yet these conditions are not analogous. Wilson has thought of the red nucleus as the center which produces decerebrate rigidity. Bazett and Penfield [5] and Pollock and Davis [6] have produced decerebrate animals in which the red nucleus was destroyed or was completely isolated above the section.

Meyers [7] has reported two cases in which the important lesion common to both was degeneration in the lenticular zone. His conception of decerebrate rigidity is different from that described by Sherrington, and consequently the clinical cases are difficult to analyze for evidence of symptoms of decerebrate rigidity. Simons [8] has reported several clinical cases, among them many cases of hemiplegia, which showed tonic reflexes. He believes that patients with an extrapyramidal syndrome and with no evidence of pyramidal tract disease never show these reflexes. The cases reported were not critically studied from the standpoint of clinical decerebrate rigidity. Brouwer [9] likewise studied

4. Wilson, S. A. K.: On Decerebrate Rigidity in Man and the Occurrence of Tonic Fits, Brain **43**:220, 1920.

5. Bazett and Penfield: A Study of Sherrington Decerebrate Animals in Chronic as well as Acute Conditions, Brain **48**:185, 1922.

6. Pollock, L. J., and Davis, L. E.: Studies in Decerebration. I. A Method of Decerebration, Arch. Neurol. & Psychiat. **10**:391 (Oct.) 1923.

7. Meyers, I. L.: Magnus and deKleijn Phenomena in Brain Lesions of Man, Arch. Neurol. & Psychiat. **8**:383 (Oct.) 1922.

8. Simons, A.: Kopfhaltung und Muskeltonus; klinische Beobachtungen, Ztschr. f. d. ges. Neurol. u. Psychiat. **80**:499, 1923.

9. Brouwer, B.: Klinisch-anatomische Untersuchung über partielle Anencephalie, Ztschr. f. d. ges. Neurol. u. Psychiat. **32**:164, 1916.

the tonic reflexes, but made no analysis of the quality of the rigidity which was present.

The case of decerebrate rigidity in man reported by Walshe,[10] to my mind meets the requirements of experimental decerebrate rigidity more closely than any of the cases so far considered in the literature. Walshe carefully studied a young woman, 23 years of age, who at necropsy was found to have a typical suprasellar cyst which contained calcareous deposits. One year previously, she complained of headaches, the onset of which were followed by sudden loss of vision. On examination, she presented marked bilateral papilledema with secondary atrophy. There were bilateral Babinski signs, with increased deep tendon reflexes and sluggish superficial abdominal reflexes. A right-sided subtemporal decompression was performed, and subsequently the patient developed a left hemiplegia. She later became semicomatose, and could neither answer nor carry out simple orders. After four weeks, she developed bilateral hemiplegia and was unable to swallow. The pulse and respirations were at all times regular and unembarrassed, while the temperature remained subnormal. She exhibited many interesting symptoms viewed from the standpoint of decerebration. The arms were held lying across the body semiflexed at the elbows, with the forearms slightly pronated and the wrists and fingers flexed. The legs were held extended and adducted, with the feet in plantar flexion. The rigidity was plastic and was typically "clasp knife" in character. The tendon reflexes were brisk and showed a tonic prolongation as well as muscle shortening after a series of rhythmically produced tendon reflexes. Plantar stimulation was followed by a flexion reflex. The tonic reflexes of Magnus and deKleijn were present, and in the article are illustrated on a model. More recently, Marinesco and Radovici [11] have reported another case that may be classed as clinical decerebrate rigidity. Their patient developed a quadriplegia and exhibited Magnus and deKleijn reflexes in addition to an extensor rigidity which apparently was quite typical of that found in the decerebrate animal. The lesion in this case is predicated as a vascular hemorrhage or thrombosis in both internal capsules, although no necropsy report is available.

I wish to report a case similar to that described by Walshe. The condition in this case was diagnosed as a suprasellar cyst and was shown by operation and necropsy to be so. While many suprasellar cysts and other lesions of the central nervous system have been observed in Dr. Cushing's clinic, this case was the only one which to my mind presented

10. Walshe, F. M. R.: A Case of Complete Decerebrate Rigidity in Man, Lancet **2**:644, 1923.

11. Marinesco, G., and Radovici, A.: Contribution à l'étude des reflexes profonds du cou et des reflexes labyrinthiques, Rev. neurol. **1**:289, 1924.

symptoms typically characteristic of decerebration. I do not believe the tonic spasms so frequently encountered in cerebellar tumors and accompanied by respiratory and circulatory irregularities are representative of decerebrate rigidity in man. It is necessary to establish the clinical picture of decerebrate rigidity conclusively and thoroughly before attempting to describe pictures of partial decerebration.

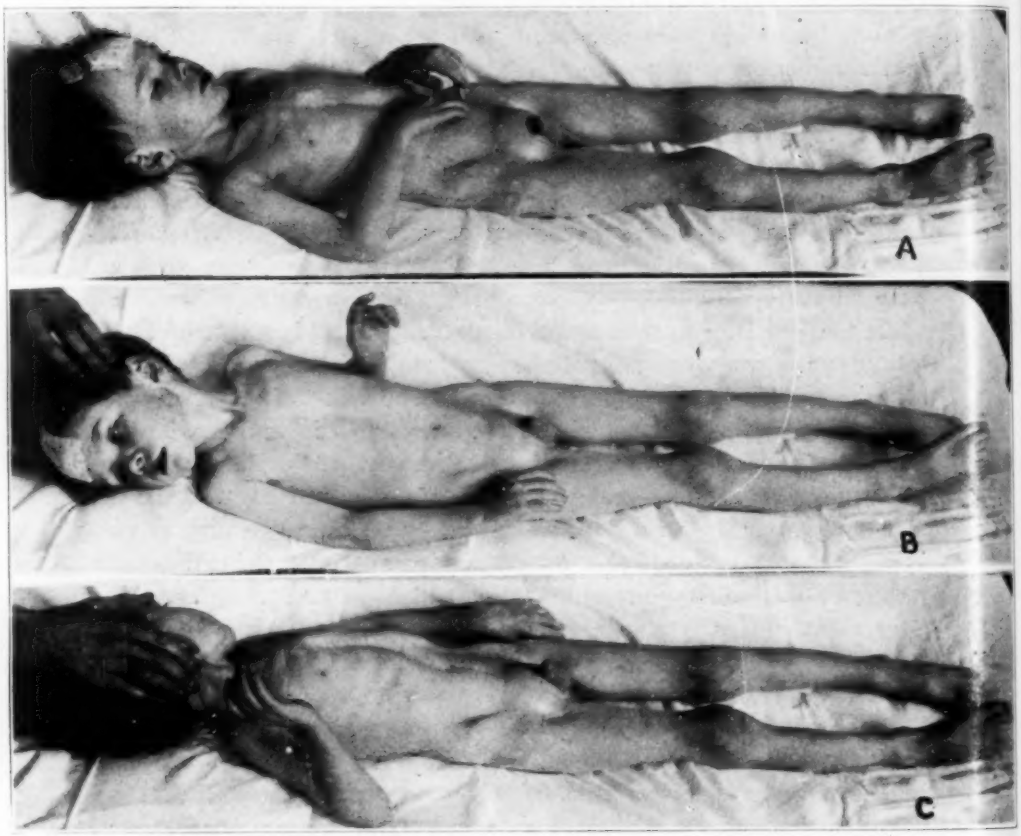

Fig. 1.—*A*, characteristic attitude of the patient; the legs were held in full extension and the arms in semiflexion; *B*, Magnus-deKleijn tonic neck reflex; chin turned to the right produced flexion of the left arm and increased extensor rigidity of the right arm; *C*, Magnus-deKleijn tonic neck reflex; chin turned to the left produced flexion of the right arm and increase in extensor rigidity of the left arm.

REPORT OF A CASE

Complete blindness, quadriplegia, headaches, semiconsciousness—preoperative diagnosis of suprasellar cyst. Partial evacuation of cyst; death. Necropsy verified a large suprasellar cyst with calcareous deposits.

Clinical History.—T. H. C., a boy, aged 4 years, referred by Dr. L. C. Heidger, Bridgeport, Conn., was admitted to the hospital, Feb. 13, 1924, with the history that at the age of 18 months visual difficulties were first noted by the parents. It was then apparent that the child could see objects in the upper visual fields much better than he could in the lower or lateral fields. At that time, he developed an internal squint in the right eye. The patient's vision gradually grew worse, until he became completely blind at the age of 2½ years. The child, however, was active and played about his home until June, 1923, when he began to complain of severe frontal headaches which recurred every second or third day and were accompanied by vomiting. He remained in that condition until December, 1923, when he became drowsy and stuporous, and his headaches became almost constant. He was placed in an asylum for the blind, where he was found and referred to Dr. Cushing's clinic. There

Fig. 2.—An area of calcification in the skull typical of a suprasellar cyst.

was nothing of note in his past history except a trauma to the head in infancy, which was not looked on as particularly serious at the time. He had never had convulsions or any other evidences of an irritative lesion.

Neurologic Examination.—On entrance, the child was lying in bed in a semistuporous condition. He was wholly unable to perform any voluntary movements with his upper or lower extremities, all of which were definitely spastic. The legs were held in strong adduction and in full extension with the feet in plantar flexion. The arms were held in semiflexion at the elbows and were kept adducted to the body. The left arm was more spastic and was held more closely adducted than was the right. The extensor rigidity was also more pronounced in the left lower extremity. The deep tendon reflexes were much exaggerated, but equally so, while all of the superficial reflexes were absent, and there were bilateral Babinski phenomena.

The neck was held quite rigid and stiff, but there was no retraction (Fig. 1 *A*). There was an absence of pupillary response to light. There was con-

jugate deviation of the eyeballs to the right. The optic disks were almost paper white, with sharply defined edges, while the retinal vessels were normal in size and distribution. Both optic cups were visible. The picture of the fundi was characteristic of a primary optic atrophy. Stimulation of the skin over the body evoked ample motor responses. At this time, the pulse averaged about 86; the respirations were 26, and the temperature per rectum was normal.

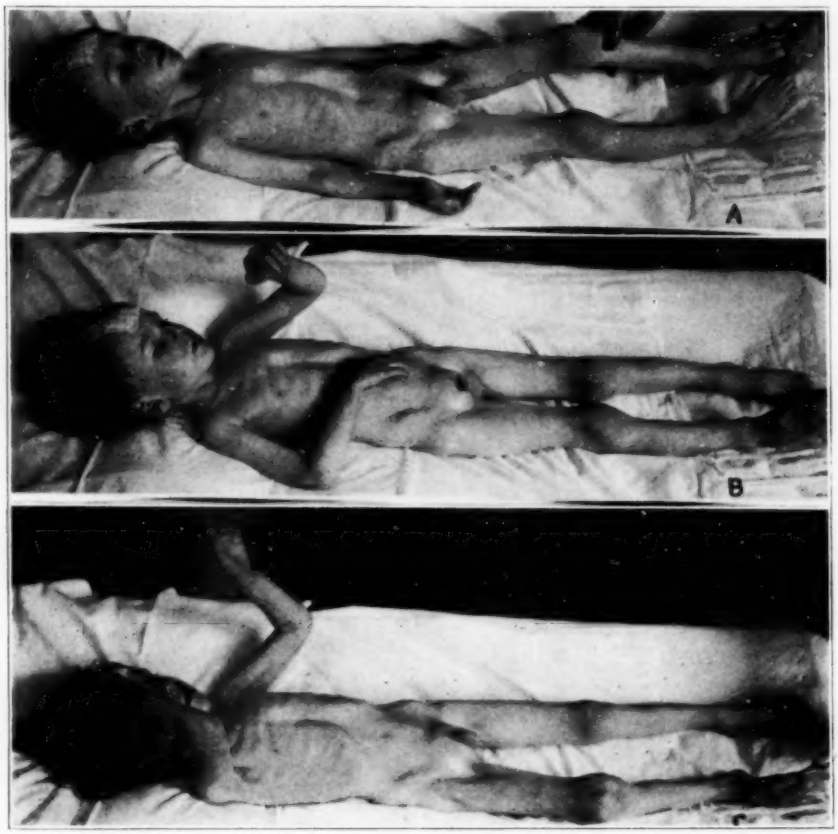

Fig. 3.—*A*, this extensor rigidity could be overcome by effort; *B*, extremities could be molded into various positions; *C*, plantar stimulation evoked a massive flexor withdrawal reflex.

Roentgen-ray examination of the skull showed the typical picture of calcification in the region of the sella turcica (Fig. 2). The roentgen-ray evidence of suprasellar cysts has been referred to recently by McKenzie and Sosman.[12]

Operation.—On Feb. 19, 1924, Dr. Cushing made a burr hole through the skull at a point usually selected for the upper angle of a transfrontal osteo-

12. McKenzie, K., and Sosman, M.: Roentgenographic Significance of Suprasellar Calcification, Am. J. Roentgen. & Rad. Therap. **11**:171, 1924.

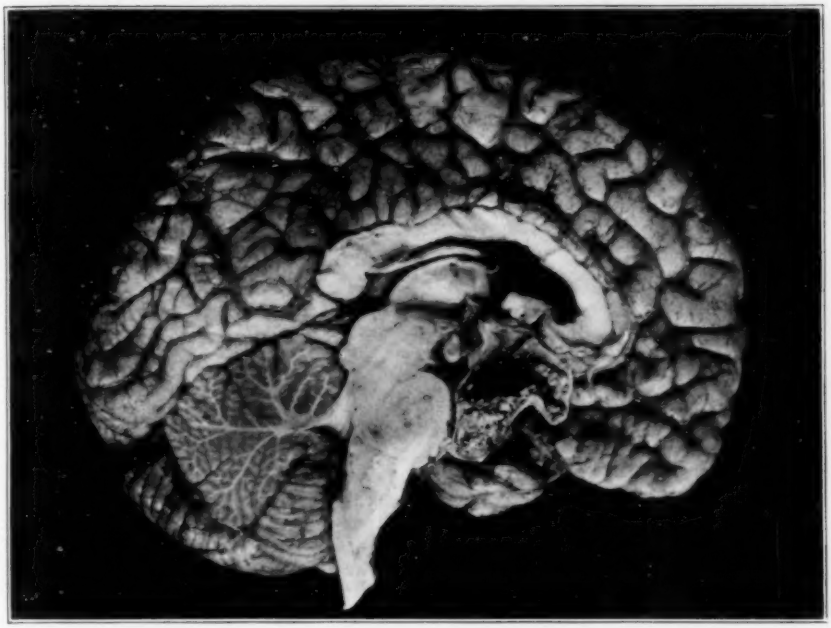

Fig. 4.—Saggital section of the brain. Size and relation to the midbrain and pons of the suprasellar cyst are shown.

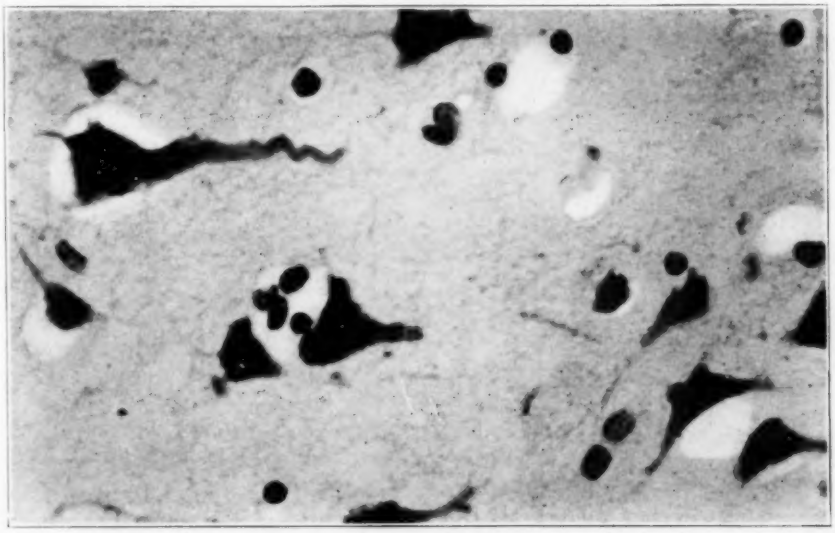

Fig. 5.—Retrograde degenerative changes in the giant pyramidal cells of the motor cortex. Bielschowsky-Plein stain; × 850.

plastic flap. A brain needle introduced toward the pituitary fossa first entered the lateral ventricle, and a large internal hydrocephalus was verified. The needle when introduced to a deeper level in the same direction struck a large cyst from which only about 15 c.c. of characteristic yellow fluid containing cholesterin crystals could be obtained.

Clinical Course.—The child was somewhat better for twenty-four hours, and then resumed his stuporous condition and gradually became unconscious. He was unable to swallow and had to be fed by an intranasal catheter.

The extensor rigidity became more pronounced in all the extremities. The rigidity is shown in Figure 3 *A*. After persistent efforts to overcome it, the rigidity would melt away in the "clasp knife" fashion. The upper extremities were held in semiflexion as described previously, but could be molded into various positions where they would remain for from five to ten minutes, after which they would gradually assume their previous posture (Fig. 3 *B*). The deep tendon reflexes were brisk and showed definite tonic prolongation with muscle shortening after a series of induced rhythmical reflexes. Plantar stimulation produced plantar flexion of the toes with a massive flexor withdrawal response at the knee and hip. This withdrawal reflex was often accompanied by movements of the upper extremities, as is shown in Figure 3 *C*. Sneezing and coughing reflexes were easily elicited. The jaws were held tightly closed and were opened with difficulty.

Magnus and deKleijn tonic neck reflexes were easily demonstrated, as is shown in Figure 1 *B* and *C*. Turning the chin toward the right shoulder produced increased extensor rigidity in the right leg with movement of the right upper extremity from the position shown in Figure 1 *A* to that of extension and pronation of the hand shown in Figure 1 *B*. At the same time, the left leg became less rigid and the left arm assumed a position of increased flexion and supination at the wrist. Similarly, rotation of the chin to the left shoulder produced increased extensor rigidity in the left leg with increase of plantar flexion; extension at the elbow and pronation at the wrist in the left upper extremity. At the same time, rigidity in the right lower extremiy became less marked, and the right upper extremity became more flexed and the wrist supinated.

The muscle tonus was at all times more marked with the child lying on its back, and he kept this position without change unless turned by the nurses. He was picked up and supported beneath the axillae. In this position, the chin fell forward on the chest and the rigidity in the extremities became much less marked, although still present. The child had no power of assuming a normal posture voluntarily, but on the other hand maintained any posture imposed on him.

The temperature taken per rectum was at all times subnormal, while the pulse and respiratory rates were regular. There was at no time anything resembling tonic fits, which invariably are accompanied by respiratory and circulatory irregularities both clinically and experimentally.

Operation.—Dr. Cushing again punctured the cyst and secured 30 c.c. of the same type of cystic fluid, in the hope that the child might improve sufficiently to justify more extensive operative procedures. The patient succumbed three days later. Forty-eight hours before death, the tonic reflexes described were not demonstrable, and remained absent.

Necropsy Findings.—The brain, hardened in situ, showed a large suprasellar cyst. Such cysts are congenital in origin and arise from the craniopharyngeal

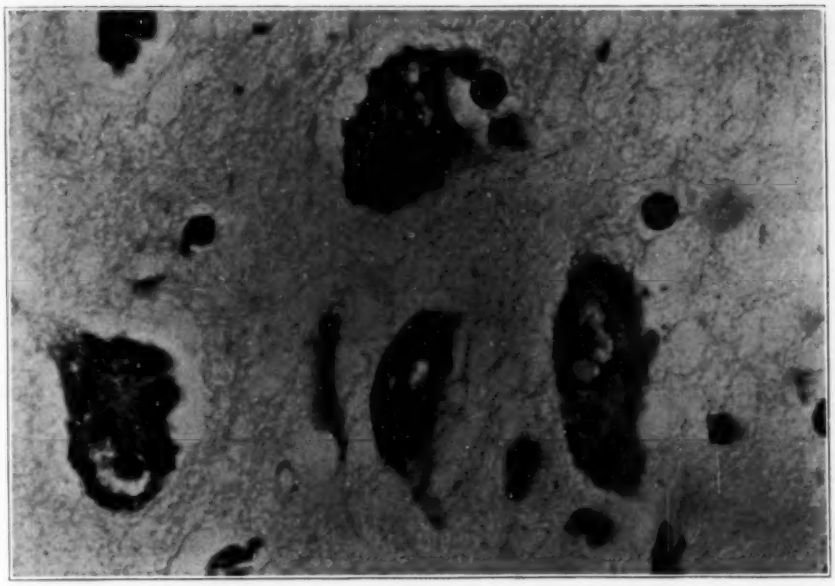

Fig. 6.—Pyramidal cells in the midbrain cephalad to the level of compression. Bielschowsky-Plein stain; × 850.

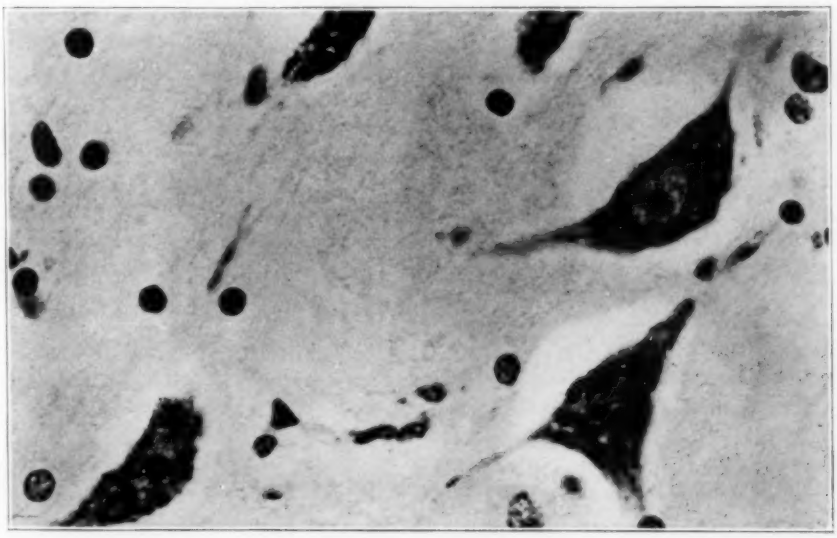

Fig. 7.—Pyramidal cells of the medulla. Note the contrast between these cells and those found cephalad to the lesion. Bielschowsky-Plein stain; × 850.

pouch. A saggital section (Fig. 4) showed the size and relations of the tumor. There were large masses of calcareous material within the cyst cavity, which produced the characteristic shadow on the roentgenogram. There can be no doubt that such a tumor was capable of producing definite and marked compression on the structures in the anterior part of the brain stem and could

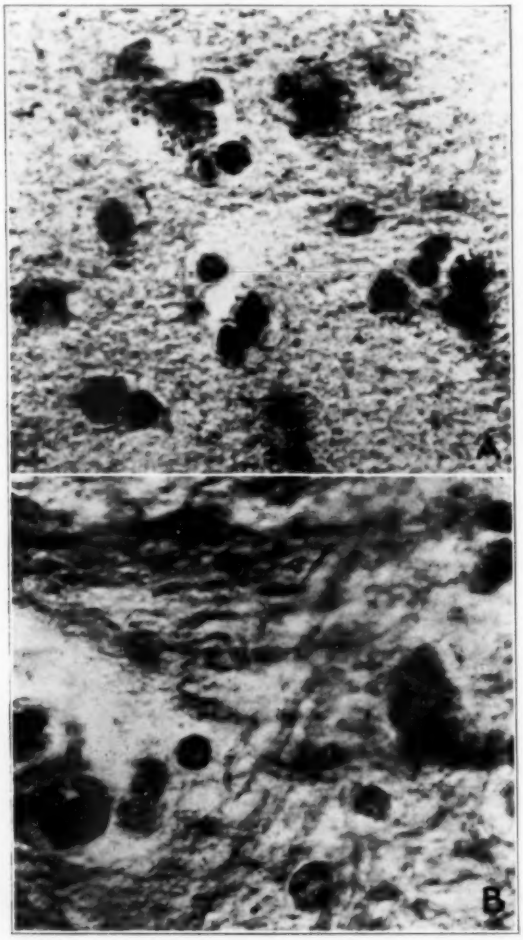

Fig. 8.—Fat droplets in the cytoplasm of the pyramidal cells. *A*, cerebral cortex; *B*, midbrain. Scharlach R stain; × 80.

produce a physiologic decerebration quite analogous to that seen in an experimental lesion through this same region.

Pathology.—Sections of the cerebral cortex from the precentral gyrus showed definite retrograde degenerative changes in the giant pyramidal cells (Fig. 5). The cells were shrunken and ill-shaped and the processes were swollen. The nuclei stained poorly and were placed excentrically. The Nissl granules were gathered in large clumps near the cell periphery and stained poorly. In most instances, they were represented by an indefinite mass which was difficult to

distinguish from the cytoplasm. The motor cells of the midbrain showed definite chromatolytic changes (Fig. 6). The cells of the basal ganglia did not show any typical changes. The appearance of the motor cells in the cortex and midbrain contrasted strongly to that of the motor cells of the lower pons, medulla and spinal cord. In these areas, typical large Nissl bodies were seen (Fig. 7).

Figure 8 shows the presence of fat droplets in the cytoplasm of the motor cells of the cerebral cortex and midbrain. No pathologic changes could be found in the neuroglia, nerve fibers or connective tissue by the Heidenhain, Walter, ethyl violet-orange G, Perdrau or Loyez methods.

SUMMARY

This case represents decerebrate rigidity in man. The suprasellar cyst produced sufficient compression upward on the brain stem to produce clinical symptoms exactly similar to the picture of experimental decerebrate rigidity. While the anatomic lesion of experimental decerebration cannot be encountered in the clinic, a physiologic lesion may occur which meets the basic requisites of such an experimentally produced decerebrate rigidity. In the nature of things, such cases are infrequently encountered.

Decerebrate rigidity is an entity by the study of which our knowledge of muscle tonus has been enriched. The application of such a physiologic entity to clinical cases should be made only after all the principles underlying that condition can be satisfied. Attention again has been called to the symptoms presented by the decerebrate animal. Although the presence of certain of the Magnus-deKleijn phenomena is not necessary, their presence corroborates the diagnosis of clinical decerebrate rigidity. The pathologic findings in the case reported would tend to show that such extensor rigidity is accompanied by definite changes in the pyramidal system cephalad to the site of the lesion. The rigidity in extension present in this case was of a character exactly analogous to that found in experimental decerebration. This rigidity was quite unlike that encountered in paralysis agitans or athetosis. Magnus-deKleijn tonic neck reflexes were also present. Irregularities of the pulse or respiration did not occur, so that there was no stimulation of medullary centers such as is frequently encountered in tonic spasms.

CEREBRAL PNEUMOGRAPHY

ITS DANGERS AND USES *

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In 1918, Dandy [1] described the procedure of injecting air into the cerebral ventricles preliminary to roentgenography. Since that time rather startling claims have been made for the localizing value of this diagnostic method. On the other hand, unexpected fatalities have made many justly cautious about its use. The difficulty of interpreting the plates has likewise called forth adverse criticism.

Discussion of the method from the point of view of risks, indications and interpretation in an attempt to determine its proper sphere in neurologic surgery is therefore much needed.

This report is based on a series of sixty cranial pneumograms, of which twenty-seven [2] are from the Neurological Institute and thirty-three from the Presbyterian Hospital. In forty-two cases, the air was injected directly into the ventricles; in twelve cases, the injection was into the spinal canal by lumbar puncture, and in six cases a neoplastic cyst of the brain was filled with it.

INTERPRETATION

Interpretation of roentgenograms depends on a knowledge of the normal variations of the ventricles both obstructed and nonobstructed. It is impossible, however, for any one to pass a reliable judgment on the plates unless he knows how the injection was made and the positions and preliminary oscillation of the head. Final conclusions should be made by one who has, in addition to these facts, a knowledge of the clinical findings as well.

The general conformation of the ventricular system and its displacement or obliteration have been well figured in the literature cited here. There are, however, a number of normal variations seen in both obstructed and nonobstructed ventricles that are of importance, but

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* From the Surgical Department of Columbia University, The Presbyterian Hospital, and The New York Neurological Institute.

1. Dandy, W.: Localization or Elimination of Cerebral Tumors by Ventriculography, Surg., Gynec. & Obst. **30**:329-342, 1920. Ventriculography Following the Injection of Air Into the Cerebral Ventricles, Ann. Surg. **68**:5-11, 1918.

2. With one exception, the patients from the Neurological Institute were not operated on by the author but by Drs. Elsberg, Taylor, Kenyon or Stookey.

which have so far been ignored. The greatest variation is seen in the outline of the posterior extremity of the lateral ventricle. Figure 1 shows a long posterior horn, whereas in Figures 2 and 3 there seems to be no posterior horn as such. The posterior end of the ventricle may be deeply notched as in Figures 4 and 5. On examining a number of normal brains, it was found that the calcarine fissure forms an elevation on the inner wall of the posterior horn, as is described in textbooks of anatomy,

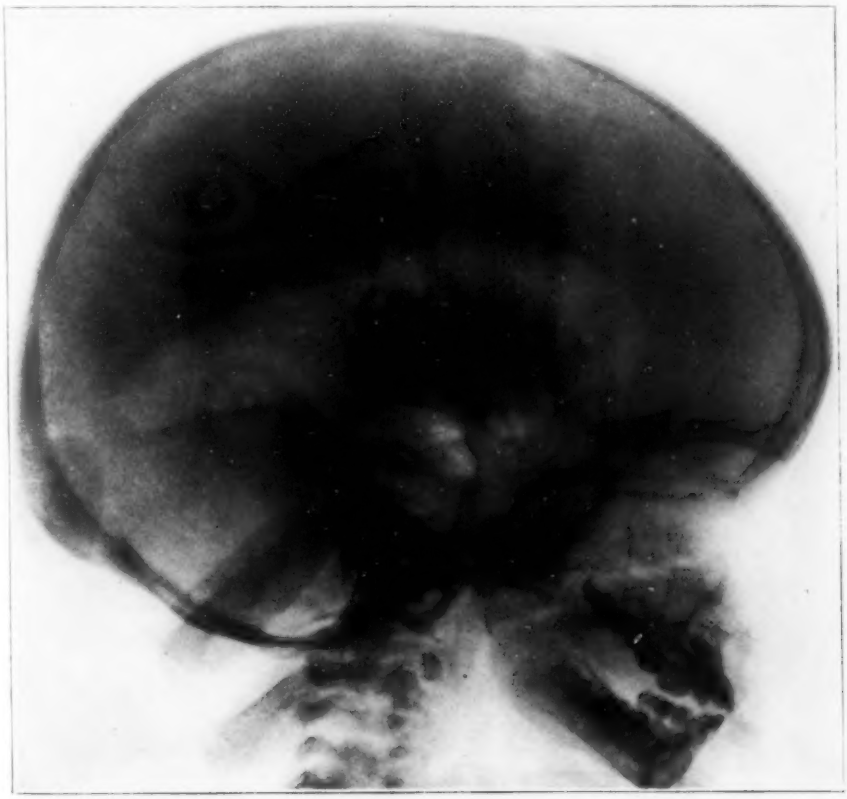

Fig. 1.—Normal lateral ventricle a little enlarged. Slender posterior horn; right side of head up. Lumbar injection (J.P.).

but that when the horn is short, the anterior stem of this fissure may cause the posterior end of the lateral ventricle to be forked in outline. Continued dilatation tends to smooth out all angles, but there may persist a definite posterior horn (Fig. 6) in the presence of marked dilatation.

In frontal lobe tumors, the posterior end of the lateral ventricle may be normal in size, or dilated, while the inferior horn on the same side as the tumor is compressed indirectly by the tumor. Figure 7 shows the

compressed inferior horn of the right lateral ventricle of another case. In this case the patient (A. R.) had a large infiltrating glioma between the frontal lobes, extending farther to the right than to the left. He died a week after exploration. Figure 8 shows the lesion.

REACTIONS

Unfavorable reactions to cerebral pneumography are of different types. In this series of sixty pneumograms, there has been no death definitely the result of the air. But one patient died thirty-six hours

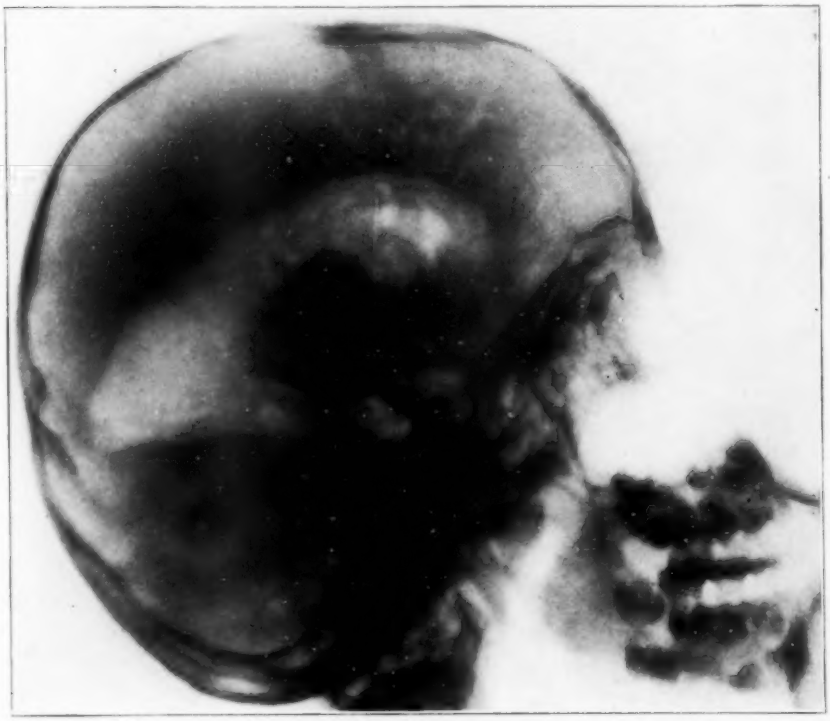

Fig. 2.—Normal lateral ventricle. Blunt posterior horn; right side of head up. Spinal injection (E.B., epilepsy).

later, for which the injection of air may have been partly responsible. B. P. gave a history of frequent dizzy spells followed by vomiting. Recently she had become unconscious in some of these attacks. Physical examination resulted in no definite localization. There was papilledema of 4 to 5 diopters. Fluid was withdrawn from the posterior horn of one lateral ventricle and replaced with air. The roentgenograms showed a moderate symmetrical dilatation of the lateral ventricles, making a presumptive diagnosis of a lesion in the posterior fossa. There

was no immediate reaction. She had a little headache that evening. The afternoon of the following day she vomited once, not an unusual occurrence. At 10 p. m., while expelling an enema, she suddenly became faint and pale. Respiration became slow and labored, and she was cyanosed. A needle was inserted to remove any remaining air, but she died at 11

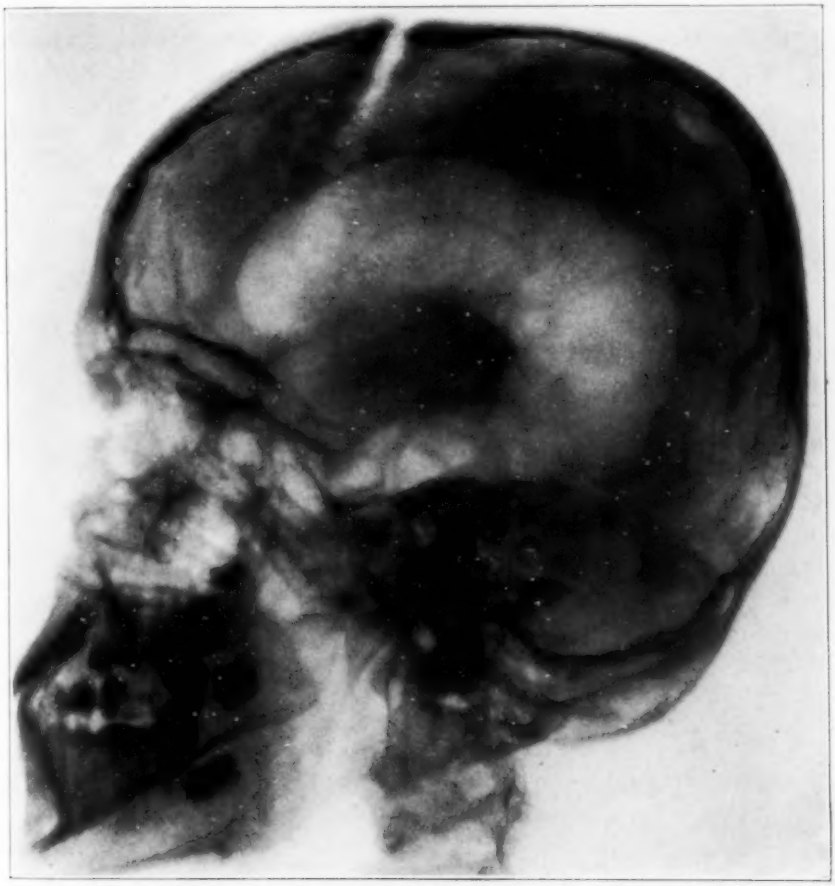

Fig. 3.—Distended lateral ventricle. No true posterior horn; left side of head up. Ventricular injection (C.S. cerebellar neoplasm).

p. m., necropsy being refused. Something happened suddenly, for which it is possible the air was a contributing cause.

There was one case of infection following air injection. This occurred in a baby that had an obstructive hydrocephalus. The temperature rose immediately after the injection, and in four or five days the baby seemed to be dying. The ventricle was tapped and fluid removed which contained many pus cells and grew a pure culture of *Staphylo-*

coccus albus. Fortunately, the block was in the aqueduct of Sylvius, and the ventricular cavities could be completely irrigated with Ringer's solution, 700 c.c. being used. The patient was much better next day. This treatment was repeated four or five times, and the temperature dropped to normal after four weeks. The baby went home in six weeks, with no evidence of infection.

In one case, E. M., the replacement of 10 c.c. of fluid from the right lateral ventricle with an equal amount of air was followed in four hours by coma and convulsions of the limbs on the same side as the injection.

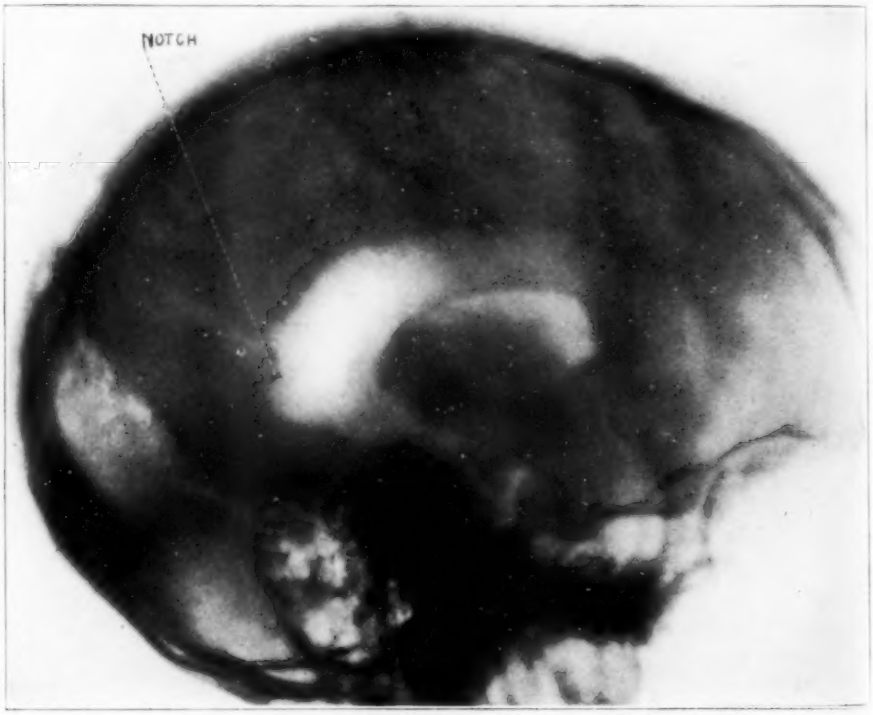

Fig. 4.—Notch in posterior end of lateral ventricle. Air can be seen in the tract made by the ventricular needle. Right side of head up. Ventricular injection (G.M., right frontal tumor).

Some or all of the air was removed, and the convulsions soon stopped and consciousness returned slowly. At necropsy, three months later, this patient was found to have an infiltrating glioma on the side of the brain opposite that into which air was injected.

In over half of the cases, replacement of cerebrospinal fluid by air is followed by a more or less moderate reaction. This may consist in a rise of temperature, which occurs any time within the first twenty-four hours, and may last up to four days. Or, the patient may give evidence

of increased intracranial pressure by vomiting, drowsiness or complaints of headache. This drowsiness at times persists as long as a week in patients who have marked papilledema.

One patient presented an alarming reaction to spinal injection of air. A boy, E. B., had a condition which had been diagnosed as idiopathic epilepsy. Fifty cubic centimeters of spinal fluid were replaced by lumbar puncture with air in small amounts. The head was elevated somewhat above the spine during the procedure, and roentgenograms showed that

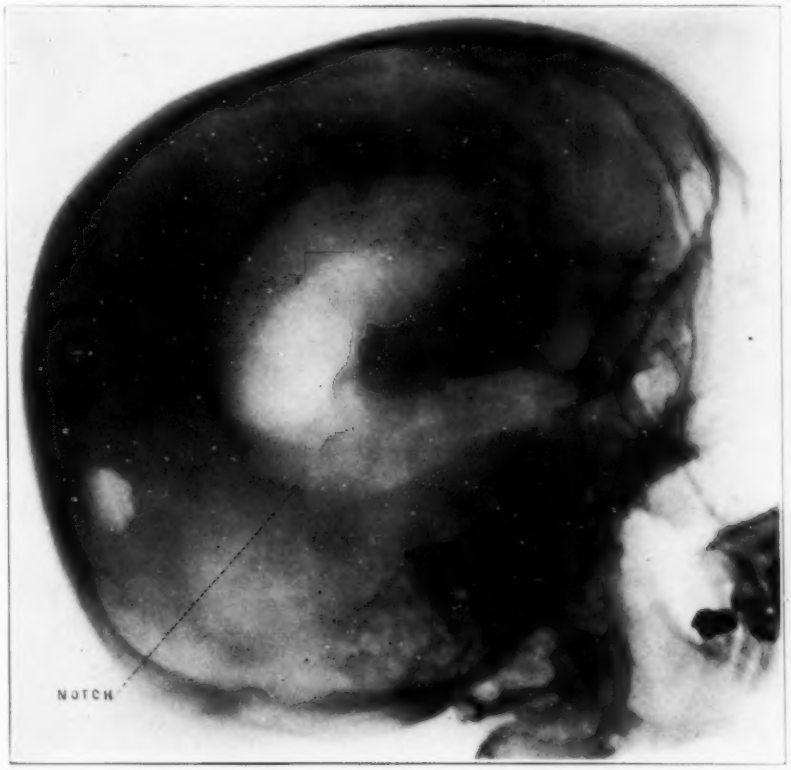

Fig. 5.—Notch in posterior end of lateral ventricle, right side up. Ventricular injection (M.T.).

air entered the ventricles and filled the intergyral subarachnoid spaces at once (Fig. 2). While in the roentgen-ray room, he vomited and became rapidly comatose. He could be roused only by painful stimuli. His pulse and respiration became very slow at recurring intervals. At these times, the heart would seem to stop and then pick up again. The abdominal and cremasteric reflexes were absent, and the plantar responses were extensor. His condition was most alarming for the first hour after the injection, and then became progressively better. His

temperature rose to 101 that night, but returned to normal in the morning, by which time the plantar responses had become normal again

A reaction of this type was never seen in cases of ventricular block. The suddenly altered superficial reflexes indicated a lesion of the pyramidal tract. The air fills the subarachnoid spaces on the surface of the brain and about the brain stem more rapidly and completely when injected by lumbar puncture than by the ventricular route. This may act as an irritant and would explain the fatalities following spinal injection where there was no ventricular block, reported in German literature. Mader [3] has reported twenty cranial pneumograms after spinal air injection in the case of infants. He reported a constant reaction more or

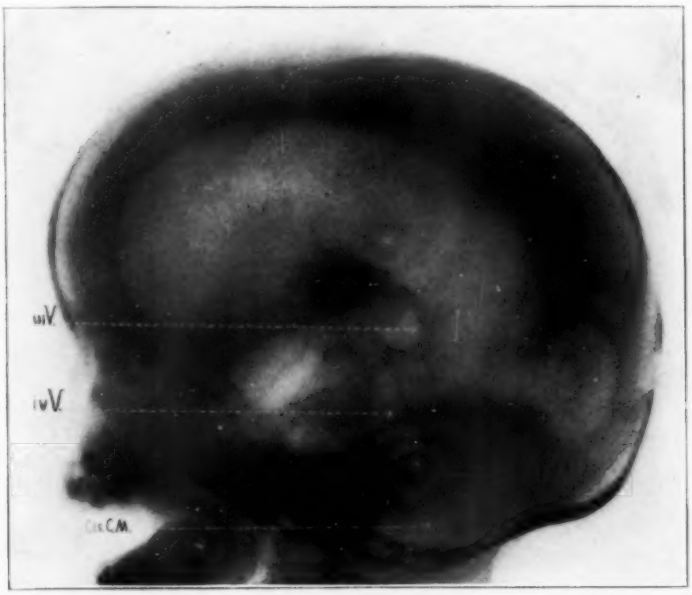

Fig. 6.—Dilated ventricular system with posterior horns preserved. Left side of head up. Ventricular injection. (C.N., hydrocephalus, obstruction in basal cisterns). *Cis C. M.* indicates cisterna cerebello-medullaris; *ivV.*, fourth ventricle, *iiiV.*, third ventricle.

less as follows: Crying followed the injection of the first air; the baby then became quiet and white; then, sooner or later, vomiting and changes in the pulse appeared. In the more severe cases, respiration ceased, and asphyxia had to be combated by artificial respiration, heart massage and oxygen administration. One of Mader's patients died. He naturally concludes that the method should be used with caution, especially during the first four weeks of life.

3. Mader, A.: Encephalographische Erfahrungen im Säuglingsalter, Med. Klin. Oct. 28, 1923, pp. 1427-1428.

That air or oxygen is irritating in the meningeal spaces is borne out also by reports of cellular reaction. Hermann [4] reported a cell count of 18,000 and 11,000 in the spinal fluid in two cases, and Mader stated that with each aspiration of fluid to be replaced by air during the procedure there were increasing numbers of small lymphocytes. We have not corroborated these findings, but we have often found the fluid blood stained toward the end of an injection.

On the other hand, when increased intracranial pressure already exists, introduction of air into the spinal canal probably affords an oppor-

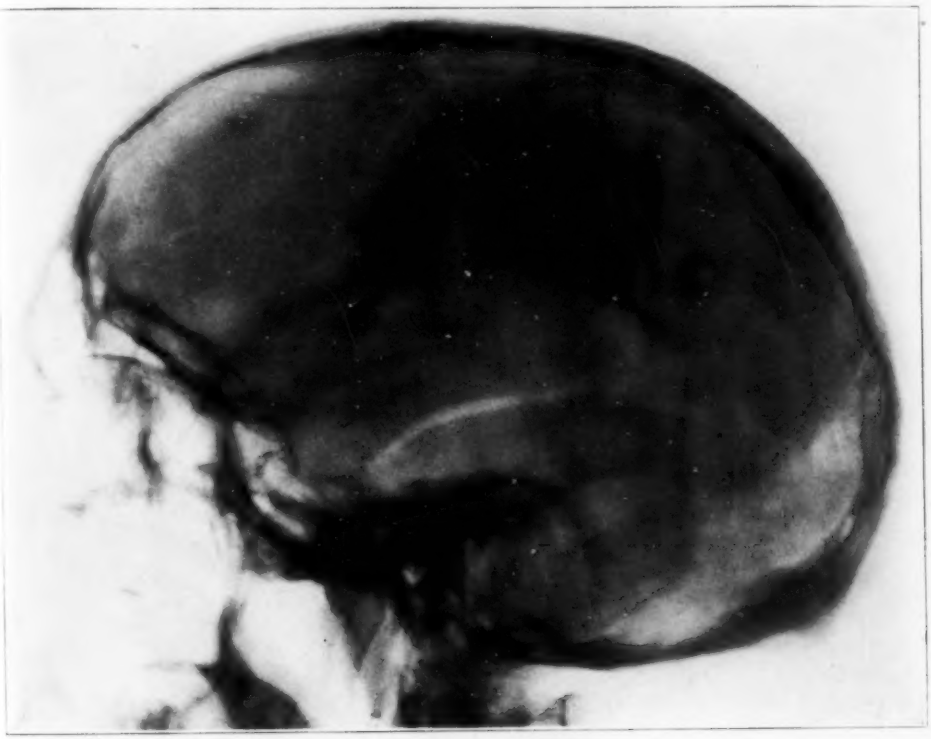

Fig. 7.—Inferior horn of lateral ventricle narrowed. Right side up. Ventricular injection. (A.R., right frontal tumor.)

tunity for herniation of the cerebellum into the foramen magnum followed by bulbar compression. It should be remarked, however, that Bingel's ingenious method of double-needle substitution is calculated to avoid this danger. The already large series of deaths, however, reported in this type of case seems to bear out the wisdom of selecting the transcerebral rather than the lumbar route of injection in the presence

4. Hermann, G.: Ueber Liquorveränderungen nach Lufteinblasung, Med. Klin. **18**:1146, 1922.

of increased intracranial pressure. Also, more information is to be gained by injecting air directly into the ventricles.

Finally, the direct replacement of ventricular fluid by air in case there is a block between the ventricular and arachnoid spaces often causes a slow increase of intracranial pressure, evidenced by vomiting,

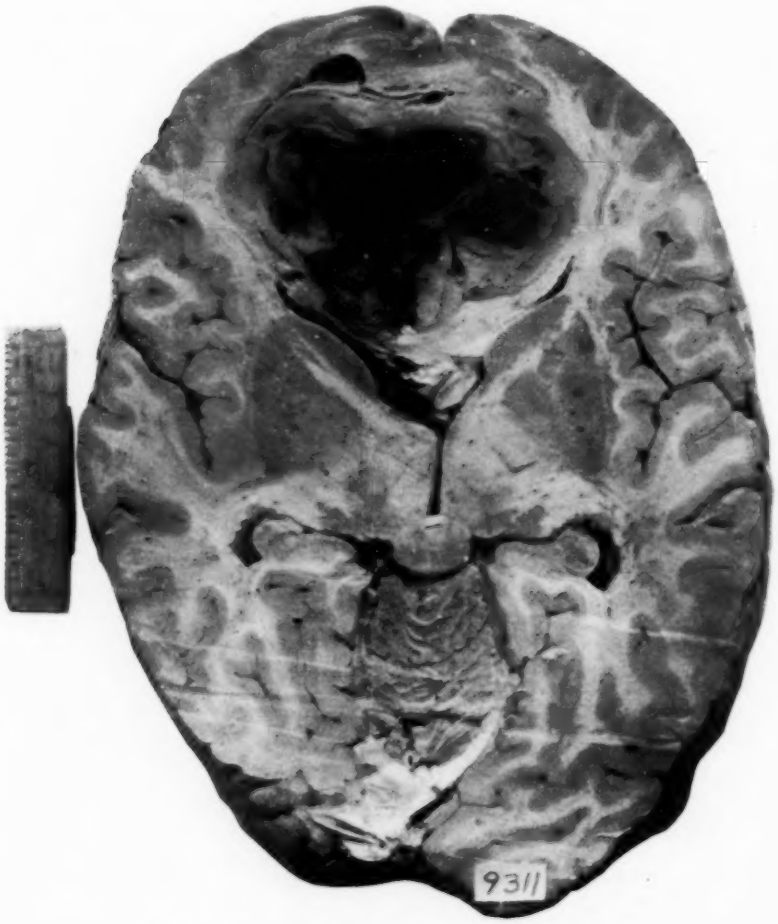

Fig. 8.—Glioma between frontal lobes (A.R.).

headache and drowsiness. If the spinal route is used, sudden death may result in this type of case. In cases in which there is no block, and especially when the air is administered by the lumbar route, there may follow evidence of bulbar paralysis which comes on quickly and disappears in a short time. This type of reaction apparently depends on the presence of a large amount of air in the pia-arachnoid spaces, includ-

ing the basal cisternae. There may be elevation of temperature with either type of reaction or in the absence of either.

In May, 1923, Bingel [5] collected reports of six deaths following the lumbar injection of air or oxygen and of three deaths after direct ventricular injection. In five of these nine cases, there was a tumor above the tentorium and in two a tumor below. One death followed lumbar injection in a case of general paralysis and one in generalized carcinomatosis with cerebral arteriosclerosis. No ventricular block was present in these two cases. Denk,[6] working in Eiselsberg's clinic, has reported three deaths, two of which were included above. All three followed injection of air into the lumbar space, while he reports thirty direct ventricular injections without fatality. His conclusion seems valid that spinal injection [7] of air is never justified in the presence of increased intracranial pressure unless preceded by ventricular decompressive puncture.

TECHNIC

Certain steps can be taken to avoid some of the reactions mentioned above. If a ventricular puncture is made, the exchange of fluid for air should be carried out with small gradual alterations of pressure. If a large amount of air has been injected, or if, while the roentgenograms are being taken there is an unfavorable reaction, the air should be removed again and replaced by Ringer's solution or the previously removed spinal fluid. On removing air from a blocked ventricle less than an hour after its injection, I have frequently found the pressure increased much above that at the end of the injection. This demonstrates that there is a rapid formation of cerebrospinal fluid, caused possibly by the irritative influence of the air. Another factor is doubtless the sudden reduction of pressure. This is borne out by the fact that after simple tapping of the ventricles in a case of obstructive hydrocephalus without introducing air, the reduced ventricular pressure is often quickly

5. Bingel, A.: Todesfälle nach Gaseinblasungen in den Lumbalkanal, bezw. in die Gehirnventrikel, Med. Klin. **19**:637-640, 1923. Die Röntgenographische Darstellung des Gehirns, Klin. Wchnschr. **1**:2191-97, 1922.

6. Denk, W.: Ueber die Gefahr der lumbalen Encephalographie bei Hirntumoren, Zentralbl. f. Chir. **12**:471-472, 1923. Die Bedeutung der Pneumoventrikulographie für die Hirndiagnostik, Mitt. a. d. Grenzgeb. d. Med. u. Chir. **36**:1, 9-28, 1923.

7. The method of intraspinal injection of air before roentgenography of the cranium was described by Dandy (Roentgenography of the Brain After Injection of Air Into the Spinal Canal, Ann. Surg. **70**:397-403, 1919) in 1919 and by Bingel (Enzephalographie, eine Methode zur Röntgenologischen Darstellung des Gehirns, Fortschr. a. d. geb. d. Röntgenstr. **28**:205, 1921) in 1921. Following the lead of the latter author, numerous communications about "encephalographie" have appeared in the German literature, the spinal route of injection generally having been used.

followed by an increase in pressure that swings beyond the original figure. In cases of ventricular block, it is wise to combat the subsequent rise of pressure by administering a hypertonic solution intravenously or a saline purgative by mouth or rectum at the close of the procedure. The possibility of causing a direct hemorrhage into a neoplasm must be borne in mind. For that reason, I have always chosen the occipital lobes for puncture after the method of Dandy, as a tumor in the occipital lobe can be ruled out by taking careful visual fields.

In general, for the four positions of the head (brow up, brow down and either side up) both a horizontal and perpendicular roentgenogram should be taken. This makes it possible to orient the bubble within the cranial cavity and demonstrate its outline in two cross sections. In the brow up and brow down position, Dr. Naffziger has well pointed out that the portion of ventricle containing air is completely filled, and the outline is therefore more reliable than in positions in which that portion of the ventricle may be only partly filled.

THE USES OF PNEUMOGRAPHY

Tabulation of the details of cases to show wherein air injection has been of benefit in each case is almost impossible, as the personal element plays so large a part. In certain clinics, the certainty of clinical diagnosis is greater than in others. In these places, it is less often necessary to use injections of air. It should be added that in some clinics the ability to interpret pneumograms makes the procedure more worth while. Where doubt exists, it may be changed to certainty by cerebral pneumography, and operation prevented or endorsed. Many of the cases in which diagnosis is in doubt prove to be deep-seated gliomas; but they may be cystic gliomas for which something can be done.

The procedure has been of service to decide that a lesion localized clinically was *not* due to tumor as the underlying ventricle was normal and exploration therefore not justified. For example: D. C., a man, aged 50, gave a story of clonic spasms of the left side of the face and left arm. The Wassermann reaction was negative. A small amount of fluid was replaced by air through a left occipital trephine hole, with local anesthesia. Roentgenograms showed the right ventricle to be patent and approximately normal in shape. The patient vomited once the following day, and had a temperature of 101 F. the night after the operation. His temperature returned to normal at once, and he did not complain of any discomfort. He went home six days after the procedure, just as he came into the hospital. In this case, exploration would have been much more dangerous, and would probably have shown a local area of softened brain. Doubtless, some neurologists would have advised

against exploration in this case, and others would have wished to give the man the benefit of the doubt.

After thorough clinical study of a case, injection of air only proves or disproves a suspicion in the mind of the neurologist. In many cases, the correct therapeutic approach might have been adopted anyway. In other cases, incorrect therapy and useless operations might have followed. Every properly made cerebral pneumogram gives information. The value of that information and its practical use are not susceptible of tabulation.

The indications for the method depend on each individual case. It should not be used as a routine in all cases in which brain tumor is suspected. Reactions to the procedure are frequent enough to contra-indicate its use when a definite diagnosis can be made by any other means. It is especially desirable to avoid the use of air when the intracranial pressure is very high. But if the diagnosis is not reasonably certain, air injection is of the greatest assistance and should prevent many negative explorations. It should therefore be used without hesitation in such cases. Its service may be twofold: first, to demonstrate the presence or absence of a tumor, and secondly, to inform the surgeon of the size and location of the ventricles, which may be of help during the conduct of an operation.

CONCLUSIONS

1. As a method of localization, cerebral pneumography demonstrates with certainty only where the tumor is not located. Final conclusions depend on a knowledge of ventricular anatomy and its variations, of the physiology of the cerebrospinal fluid and the clinical aspects of the case.

2. Reactions to the method are in general of two types: first, an increase of intracranial pressure (most to be feared in cases of marked papilledema); second, evidence of bulbar irritation or paralysis. The former may be long continued; the latter is immediate. In over half of the cases, hyperpyrexia follows the injection for from one to four days.

3. Certain provisions can be made to combat reaction, such as removal of the air, the use of hypertonic solutions, etc.

4. Ordinarily, a horizontal and perpendicular roentgenogram should be taken with the head in each of the four possible positions. Systematic oscillation of the head should precede each new position.

5. Finally, cranial pneumography in properly selected cases is of the greatest assistance in localizing tumors and outlining the ventricles before operation. As a procedure it must in no wise diminish the thoroughness of neurologic examinations and should not be employed unless these examinations prove indeterminate.

BEHAVIOR OF THE PLANTAR REFLEX IN JACKSONIAN EPILEPSY

WITH SOME OBSERVATIONS ON THE PATHOLOGIC PHYSIOLOGY OF THE BABINSKI SIGN

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In the complete description of the toe phenomenon and its diagnostic value, which Babinski gave in 1898 when reviewing the various diseases in which this sign might be observed, he said:

> In a case of partial epilepsy, I have had the opportunity to observe the following phenomenon. The patient was a man subject to attacks of jacksonian epilepsy, in which the convulsive movements occurred on the left side of the body. Having examined the patient immediately after an attack, I observed that stimulation of the sole of the foot gave rise to extension of the toes on the left. When this test was tried while the patient had no attack, the cutaneous plantar reflex was normal, and the left side presented no disorder of motility.

In 1899, Babinski added further observations concerning the toe sign in what is known as idiopathic or essential epilepsy. He said that in persons whose cutaneous plantar reflex had been normal when no attacks were present, he had observed this sign at the moment of an attack or after the attack had ceased for a more or less extended lapse of time. The phenomenon of the toes may therefore be unilateral or bilateral. It is at times accompanied by increased activity of the tendon reflexes with ankle clonus and abolition of the anal reflex.

In the description of convulsive attacks, these ideas, so far as concerns the plantar reflex, have generally been restated in treatises of medicine and neurology, all neurologists having had the opportunity of confirming them.

In recent years, I have had the opportunity of observing the behavior of the plantar reflex in two cases in which jacksonian fits have occurred which has not conformed to the classical description given by Babinski.

REPORT OF CASES

Case 1.—A woman, aged 64, had had several attacks of loss of consciousness, accompanied by a number of convulsive movements, one of which occurred in September, 1923. Mild signs of defective function of the pyramidal tract were present on the left: the sign of the platysma, reflex abduction of the fifth toe (fan sign) without extension of the great toe, mild hypotonia of the arm, and a slight increase of the tendon reflexes in both the upper and

lower limbs. The organic cause of these attacks could not be established by an examination of the blood and spinal fluid, or the eyes, all of which were negative.

On Jan. 24, 1924, after an aura, convulsive attacks appeared on the left side in the face and limbs. The examination began after the cessation of the first series of attacks. The patient had not regained consciousness completely. The face was normal; the upper limb was flaccid and inert; the lower limb showed normal tone. These observations were scarcely finished when a new attack began, which commenced with twitchings of the mouth on the left and with conjugate movements of the eyes toward the left. Then the twitchings extended to the upper limb and showed slightly in the lower limb. These movements continued for several minutes, and then the attack ceased. The face returned to repose. The upper limb remained inactive and flaccid for several minutes, while the lower limb showed normal motility as soon as the patient recovered consciousness.

During the entire duration of the convulsions the plantar reflex was examined repeatedly and at as short intervals as possible. The reflex was never abolished or even enfeebled, and both on the left and right the reaction was always normal flexion. After the attack, the plantar reflex continued to be normal.

Case 2.—A man, aged 62, a factory worker, was the victim of an accident on Nov. 17, 1922. While engaged in pushing a small truck, he struck his head violently against a beam, as a result of which he had a contusion above the left orbit. After a short period, during which the patient noticed nothing except local discomfort and pains in the head, there appeared some clouding of intellect, a return of headache, and vomiting. This condition finally became more severe, and he passed into coma, interrupted by convulsive attacks.

It is most probable that the injury caused at the same time a fissure fracture of the skull in the region of the orbit and an extravasation of blood into the cranial cavity. Indeed, the spinal fluid was tinted with blood, which color remained after centrifugalization, and presented all the reactions of blood.

At a neurologic examination in the evening of Dec. 4, 1922, he was in a state of coma. The hands and face were in repose, not rigid or paralyzed. The tendon reflexes were normal in the upper and lower limbs on the left, with the exception of the Achilles reflex, which was weak. The limbs on the right side showed mildly diminished reflexes. The abdominal and cremasteric reflexes were abolished.

The plantar reflexes were in extension on the right and left, the reaction being particularly vigorous on the right. There was no ankle clonus.

In the course of the examination, a convulsive attack occurred, the ninth during the day. The patient, inert at this time, gave a sort of groan, and then began to close his right fist, which began to move away from the trunk. The forearm was in fixed pronation. At the same time, the face and the eyes deviated strongly toward the right and showed violent convulsive movements. Less active twitching was present in the right upper extremity, while the lower extremity became a little stiff, with mild displacement of the foot and flexion of the toes. The limbs on the left side were somewhat stiff, but showed no twitching.

This convulsive phase was but of short duration, and soon the limbs relaxed, those on the right side being for a short time mildly paralyzed. After returning to rest, a second attack occurred.

During the entire duration of these two convulsive attacks and the period which followed them, the plantar reflexes were examined at short intervals.

In all examinations, the results were the same. As soon as the rigidity which held the toes flexed had ceased, plantar stimulation, which had previously produced a Babinski sign both on the right and left, caused no reaction. Only after a lapse of time after the foot had been in repose did the Babinski sign reappear on both sides.

COMMENT

The plantar reflex may therefore behave in various ways in the course of jacksonian epilepsy.

1. The plantar reflex may be in flexion between the attacks and remain in flexion during the attacks (Case 1).

2. The plantar reflex may be in flexion between the attacks and show extension at the end of the attack (original observations of Babinski).

3. The plantar reflex may be in extension between the attacks and be abolished temporarily after the begining of the attack (Case 2).

It may be possible that there are cases in which the reaction in extension persists during and after the attack. It would also be important to discover whether abolition of the normal plantar reflex occurs in certain cases.

In any event, in the articles and treatises, these changes of the plantar reflex have not been commented upon. The only observation which has been made referring to abolition of reflexes refers to the abolition of the anal reflex. The temporary weakening of the tendon reflexes is also commented on.

Since it is true that in the course of the attacks the plantar reflex is not always affected as classically described, it seems reasonable to attempt an explanation of the absence of the sign of Babinski in Case 1 and its disappearance in Case 2.

We may ask first whether when the Babinski sign appears following convulsions it is not due to fatigue of the cortical centers and of the motor tract with release of the lower centers controlling extension. Its failure to appear in Case 1 may be due to the stimulation being most marked in the centers for the face and the upper limb. Not being diffused to the centers of the lower limb, the results of exhaustion of the cortex, easily seen in the arm, were not apparent in the leg.

On the other hand, in Case 2, the reaction of extension, which depends on the lower centers released by the cortical exhaustion, may momentarily be annihilated by the fact that the stimulation traveling down the motor tract transitorily maintains its predominence. The following observations of Kinnier Wilson throw further light on this question.

As for the influence of the cortex on all this complex potentiality of movement at mesencephalic and at spinal levels, we fortunately possess some experiments of Graham Brown which are, in my opinion, of the first importance

and significance, and which appear not to have received the attention they deserve. In the decerebrate ape, Graham Brown produced the usual mesencephalic reactions already discussed, say postural flexion of the left upper extremity. While this was persisting, the appropriate crus (corticospinal tracts) was stimulated. Immediate augmentation of flexion occurred; after withdrawal of this crus stimulus, one might expect the mesencephalic flexor after-discharge to remain unimpaired. But this is not the case. On the contrary, the postural after-discharge vanishes immediately, just as after an ordinary pyramidal stimulation. In other words, stimulation of the corticospinal system "wipes out" the existing postural reactions derived from stimulation of the non-pyramidal system. In Graham Brown's words, "non-postural cerebral activity seems to abolish postural midbrain activity."

The significance of this will at once be obvious to the reader. At any moment cortical, voluntary, activity obliterates that of lower motor centers; rapid, phase changes are possible because of the master control of the cortex, the actual nature of the activity of which has now received physiologic demonstration.[1]

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1. Wilson, S. A. K.: The Old Motor System and the New, Arch. Neurol. & Psychiat. **11**:385 (April) 1924.

SPECIAL ARTICLE

THE ANATOMY, CLINICAL SYNDROMES AND PHYSIOLOGY OF THE EXTRA-PYRAMIDAL SYSTEM *

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The subject under consideration is a rather difficult and extensive one, and I agree with most of the authors, especially S. A. K. Wilson, that nearly all problems in connection with it are still unsolved. I will, however, attempt to explain my own conception of the extrapyramidal system and of its lesions.

DISORDERS OF MOTILITY

It is well known that *all extrapyramidal diseases have this factor in common—they all present characteristic disturbances of motility associated with changes in muscle tonus.* These disorders of motility can be definitely differentiated from the pyramidal tract, and three forms can be distinguished, all of which are manifested in the course of voluntary, automatic and reflex movements. These three forms are: (1) hypokinesia; (2) diminished or increased muscle-tone states, manifesting themselves in either hypotonia or hypertonic rigidity; (3) hyperkinesia.

1. The *hypokinesiae* consist of failure of innervation during voluntary movements (mild paralysis), consisting of a slowing of the beginning of the execution and of the excursion of a movement with easy fatigability. Then there occurs a failure in the automatic physiologic association which, under normal circumstances, complete and "round out" voluntary movements. There also occurs a peculiar lack of reactive movements which follow reflexly all afferent sensory stimuli. (Such movements are: movements of defence, protection, flight, fear, automatic orientation, arrested attention, movements of expression, and gestures.) Finally there occurs a failure of "assisting" movements—movements that usually accompany automatically every purposeful act, such as sitting, standing, walking, chewing, swallowing and talking. These are movements that insure an undisturbed synergy in all acts of motility.

2. The disturbances of muscle tone, i. e., the state of muscle tension which the entire skeletal musculature assumes without any voluntary innervation during rest, attitude and position, may appear in the form of hypotonia, as

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* This paper constitutes part of my lectures on neurohistopathology, given under the auspices of Columbia University, New York (April 7 to May 1, 1924), and is a complete description of my lecture given, May 13, before the section of Neurology and Psychiatry, New York, and before the Section on Medicine, College of Physicians of Philadelphia on May 19, by invitation. It is a review of my book "Die extrapyramidalen Erkrankungen mit besonderer Berücksichtigung der Pathophysiologie der Bewegungsstörungen" (Julius Springer, 1923) which contains the literature and many illustrations.

in chorea or in the form of rigidity of individual groups of muscles. This form includes the increase in the permanent, plastic, form-giving muscle tonus commonly designated as rigidity which accompanies increased passive resistance to muscle stretching, delayed muscle "after-contraction," adaptation and fixation tension, with the peculiar anomalies of posture and attitude. All these phenomena of rigidity or hypertonicity play a significant rôle in the cataleptic attitudes of these patients, in adiadokokinesis, in propulsion and retropulsion, as well as in voluntary movements where they give rise to a slow incomplete execution and repetition of movements. Finally, we have here the various forms of tremors and shaking phenomena of disturbed coordination which are apparently due to improper innervation of the antagonists.

3. The *hyperkinesiae* appear in the form of the well-known choreiform and athetoid motor restlessness, torsion spasms, hemiballismus, tics and myoclonias. Chorea and athetosis are to be regarded as associated and expression movements consisting of two components: (*a*) the increased or hyperkinetic and (*b*) dissociation or ataxic component.

Choreiform hyperkinesia is a distorted and exaggerated physiologic movement and parakinesiae are involuntary complicated movements which resemble the voluntary expression movements. Athetosis also is a distorted and at the same time exaggerated associated and expression movement, in which the disruption of the lower tonic and static functions is predominant. We believe that the manifestations of torsion spasm and torsion dystonia form a separate subtype of athetosis. *Torsion dystonia* is characterized by the presence of irregular, arrhythmic convulsive movements, resembling involuntary movements, in extensive parts of the body, during which extreme twistings (torsions) of the trunk, spinal column and proximal ends of the extremities are the predominating features. Spastic torticollis with its—at times—rhythmic and at other times irregularly appearing spasms of the muscles of the neck, is a partial manifestation of torsion dystonia. The term *hemiballismus* is employed to designate the uncontrollable, incoordinated, twisting, massive movements involving an entire half of the body with an exquisite tendency to twist and roll the body. Myoclonia and tic closely resemble chorea, but are limited to individual muscles or muscle groups.

Clinically we can distinguish three syndromes: (1) the choreiform, (2) the akinetic-hypertonic (Parkinson) and (3) the athetotic. The diseases belonging to these three syndromes have as their anatomic basis an affection of certain definite parts of the basal ganglions. The entire gray substance in this anatomic region is designated the extrapyramidal system, and the motor disturbances due to disease of these centers are called extrapyramidal motor disturbances. But since in addition to the extrapyramidal system as represented by the basal ganglions there also exists a second mechanism subserving extrapyramidal motor coordination in the form of the fronto-ponto-cerebellar system, we designate the former as the principal extrapyramidal system. Of course, designation is only for the sake of brevity and is not meant to imply that the other extrapyramidal system is not as important as the principal one. Here, however, we shall limit ourselves to the latter.

The extrapyramidal system consists, in its main subdivisions, of the following gray nuclei: (1) striatum (nucleus caudatus plus putamen), (2) pallidum, (3) corpus Luysi and (4) substantia nigra Soemmeringii. The red nucleus, too, indirectly belongs to this system. A study of the anatomy of the whole system, especially the interrelations of the single centers, reveals the following facts: The striatum, which represents as its main subdivisions the nucleus

caudatus and the putamen, is a genetically and morphologically uniform mass, developed out of the same matrix as the cerebral cortex (alar plate of the prosencephalon), and is essentially also constructed like it. In its cell structure, it shows two types of ganglion cells—infrequent large ganglion cells among numerous smaller cells. This nucleus derives its stimuli from special zones in the thalamus, and its outgoing fibers terminate in the pallidum. Only, with C. and O. Vogt we have to assume that the afferent fibers terminate in the vicinity of the small ganglion cells and that these then exert their influence on the large long axon cells and from these the striofugal fibers originate and transfer the stimulation to the cells of the pallidum. The striatum thus appears to be a highly developed regulating organ endowed with cortical characteristics and superior to the pallidum, i. e., a special cortex to the pallidum.

The pallidum is a simply constructed gray nucleus, which must ontogenetically be assigned to the hypothalamic portion of the diencephalon; it is developed out of the ground plate portion of the prosencephalon—contains only one kind of ganglion cell. The pallidum is rich in fibers which appear to be of a variety of types: (*a*) afferent types coming from the same thalamic areas as the afferent strial fibers and (*b*) fibers from the striatum itself. The latter are extremely complicated and numerous.

Pallidofugal fibers connect with particular zones of (*a*) the thalamus and (*b*) hypothalamus. The latter consists of numerous fibers to the capsule of the red nucleus, to the corpus Luysi, to the substantia nigra, to the nucleus of the posterior commisura and to the oral part of the pons.

The corpus Luysi or subthalamicum has ontogenetically the same matrix as the pallidum and has a ganglion cell structure similar to that of the pallidum. It is an almond-shaped, sharply delimited gray nucleus of the hypothalamus, deriving its chief afferent fibers from the thalamus, especially from the pallidum, and sending numerous fibers to the capsule of the red nucleus and into the substantia nigra.

The substantia nigra is a macroscopically visible gray mass, lying at the foot of the pes pedunculi, divisible into two differently constructed zones: (1) a zona compacta lying dorsally and provided with characteristic large ganglion cells containing melanin and (2) a zona reticulata lying under it and constructed like the pallidum. The substantia nigra is the only one of these centers that possesses direct cortical fibers coming from the centralis anterior, operculum and the posterior part of the frontal lobes. The substantia nigra also contains richly developed connections with the pallidum, corpus Luysi, the red nucleus and the thalamic nuclei. Descending fibers extend from it to the pes pedunculi, the anterior part of the corpora quadrigemina and geniculata and the lemniscus area of the tegmentum-pontis.

The nuclei of the posterior commisura, nuclei Darkschewitschi and interstitialis receive their impulses from the nuclei of Deiters and Bechterew and give origin to the posterior longitudinal bundles (Fig. 1). The posterior longitudinal bundle constitutes that phylogenetically old connecting and reflex tract which is always encountered at the same location, and which, extending through the whole brain stem, descends into the cervical cord (anterior horn). It establishes interrelations between the nuclei of the eye muscles, and brings the vestibular apparatus into relation with the pallidum, the nuclei of the eye muscles and the spinal cord. It is to be regarded as the important coordinating tract of the whole motor apparatus of the midbrain and brain stem, including the nuclei of the eye muscles.

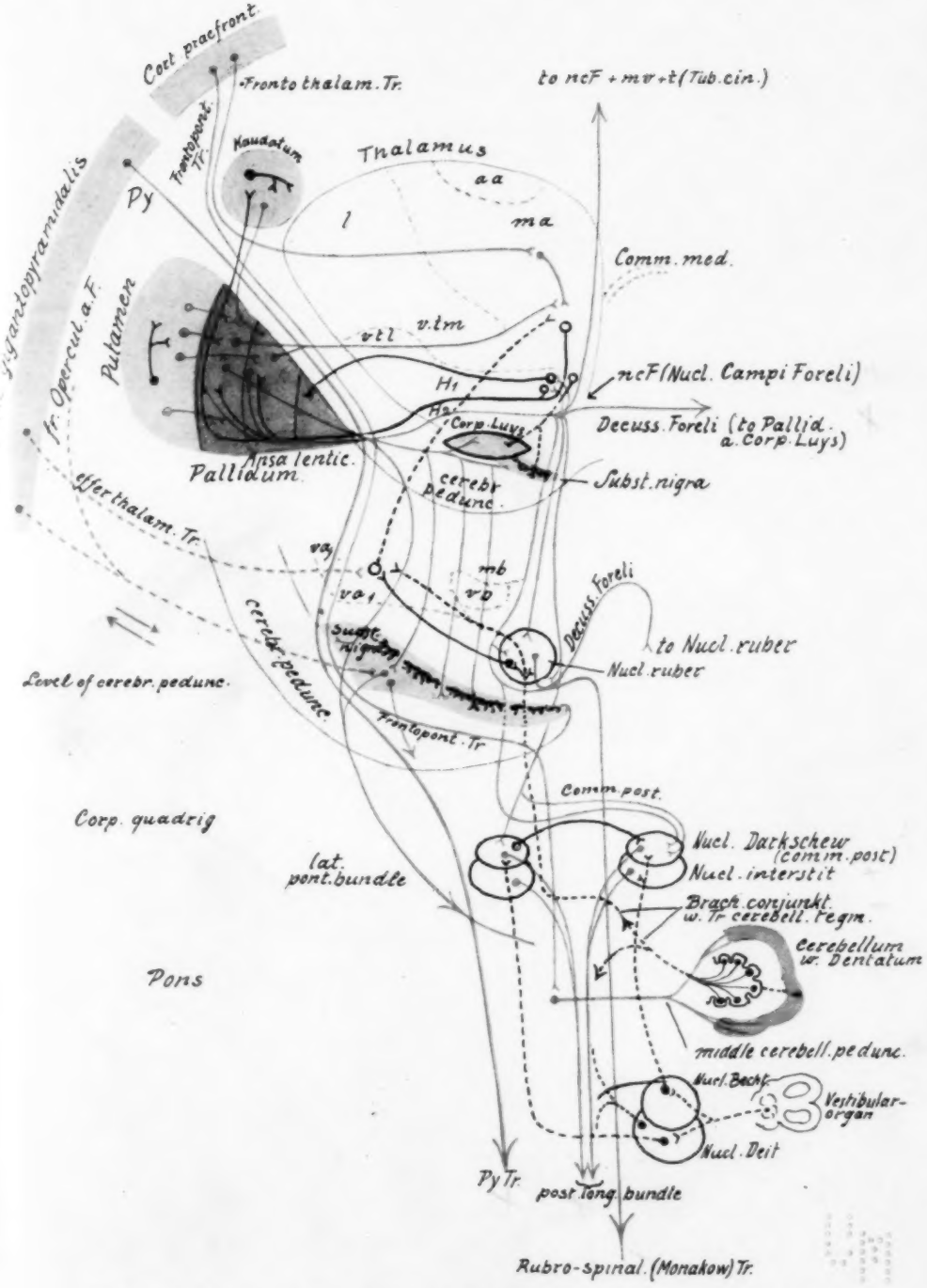

Fig. 1.—Schematic representation of the anatomy and interrelations of the extrapyramidal system (modified after C. and O. Vogt). The black lines indicate the afferent tracts to the extrapyramidal system, especially the striopallidum; the green lines, the efferent tracts; the red lines, the pyramidal and fronto-pontocerebellar tracts; the interrupted lines, the efferent thalamus tracts, the cortical connections of the substantia nigra, the dentato-rubral tracts and the fibers from Deiters' system.

In the tegmentum of the midbrain and brain stem we have, as is shown by the valuable experimental work of Magnus and his school, a motor apparatus of great importance both for animals and man. This apparatus seems to be connected with the posterior longitudinal bundle, and when stimulated proprioceptively controls, by virtue of the manifold reflexes of position and station, the harmonious distribution of tone throughout the body musculature, and regulates the precision of motions and positions. This powerful coordinating mechanism operates, according to the experimental results of Magnus, largely independently of the cerebellum and the higher cerebral centers. This is true not only in lower animals, but even in monkeys.

The red nucleus, the fibers of which are elaborately and intimately bound up with the pallidum as well as the corpus Luysi and substantia nigra, is to be regarded as the oral hypertrophy of this whole motor apparatus, the latter apparently consisting of several superimposed layers (Fig. 2). It is

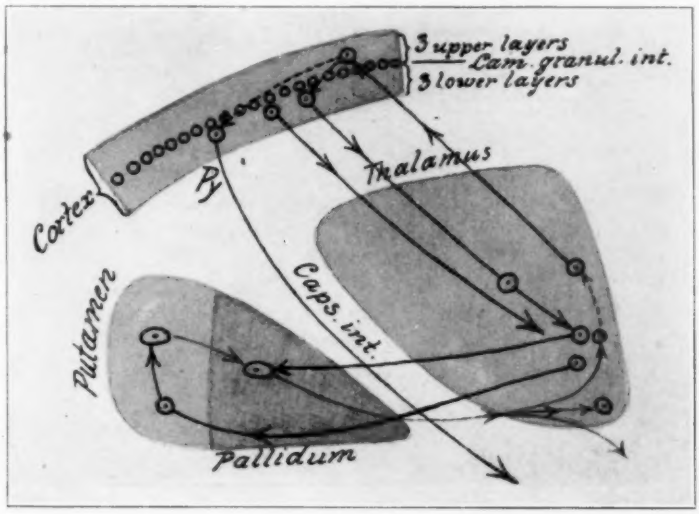

Fig. 2.—Schematic representation of the thalamostriopallidal interrelations and the thalamocortical connections. This scheme also shows how the cortical influence is exerted on the extrapyramidal system through the thalamus.

the most important organ of tone in this group, and must be regarded as playing an important rôle in the function of posture and as the special center for normal tone distribution. The rubrospinal tract of von Monakow, originating in the magnocellular portion of the red nucleus and crossing mostly in the decussation of Forel at the level of the red nucleus, is the efferent tract for these functions to the anterior horns of the spinal cord.

The red nucleus is also the chief terminal station and passageway of the dentatum tract, and is, therefore, an efferent cerebellar nucleus. As such it carries impulses from the cerebellum to the coordinating mechanism of the midbrain. In the higher animals we find, in addition to the large cells, a portion provided with small cells, intimately connected with the frontal lobe and increasing in complexity as we ascend the animal scale and reaching its maximum in man. The red nucleus is therefore a projection center for the cerebellar impulses to the frontal lobes.

Finally, the red nucleus by virtue of the highly developed pallidorubral and corpus Luysi-rubral fibers, also serves as an important connecting nucleus between the striopallidum and corpus Luysi and the motor coordination apparatus of the brain stem.

This motor coordination apparatus of the midbrain and brain stem, together with the red nucleus—as the center of all reflexes of tone, station and posture—are therefore under a double influence and stabilization (Sicherung): on the one hand, under the cerebellar influence through the dentatorubral tract, on the other hand, under the influence of our extrapyramidal system.

It is evident that the consideration of these anatomic and physiologic facts is of utmost importance for the elucidation of the complicated conditions of human pathology.

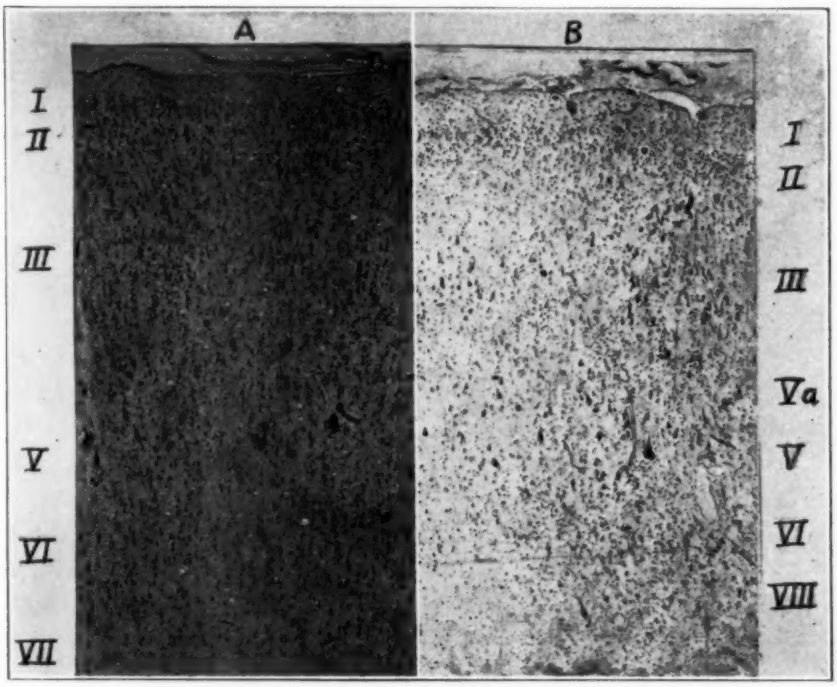

Fig. 3.—Centralis anterior (*A*) normal; (*B*) in Huntington's chorea. Nissl stain photomicrographs showing the atrophy of the cortex and the appearance of a glious pseudonuclear layer (*Va*). The Roman numerals indicate the cortical layers of Brodmann.

The following fact is of equal importance: The efferent pallidal fibers run principally to the lower centers of the homolateral side, but partly also to the contralateral side. Thus we have a bilateral extrapyramidal innervation, which through the crossing of von Monakow's bundle innervates mostly the contralateral side.

It must be borne in mind that the pathologic process in the human being, for reasons not as yet clearly understood, behaves differently from the physiologic phenomena observed in animal experimentation by the Magnus school and others. So we see that in the lower animals—the only material that can

be employed in the Magnus experiments—the removal of the centers lying orally to the red nucleus produces no characteristic changes in tonus or disturbances in locomotion, just as the experimental destruction of the substantia nigra region in animals results in no marked interference with tonus and postural function. On the other hand, injury to the extrapyramidal centers in man is accompanied by grave disturbances in tonus and in movements of an extrapyramidal character.

A study of the more or less regularly occurring changes in these gray centers and the extrapyramidal syndromes and maladies resulting, yields the following data:

CHOREA

The chorea syndrome is seen in its purest form in *chronic progressive chorea, Huntington's disease,* with the demonstrable hereditary influence, as well as that form which resembles it clinically but has no demonstrable heredity or known etiology. Both these forms are, in my experience, related to definite anatomic and histologic findings. Pathologically, chronic progressive chorea has as its basis a diffuse and purely parenchymatous degenerative process extending over large areas of the central nervous system—and showing predilection for a particularly serious degeneration in certain definite parts. It, thus, invariably finds its main localization in the striatum, especially in the caudate nucleus, resulting in a marked degeneration and eventual disappearance of the small ganglion cells. Ramsay Hunt was the first to recognize this fact.

I have had the opportunity to examine anatomically an "abortive" case of Huntington's chorea of two years' duration, in which the movements were more those of parakinesis than of chorea. In this case, I found a definite but milder degeneration of the small ganglion cells of the striatum. We can say, therefore: In milder affections of the striatum more complicated parakinesis closely resembling normal gestures may predominate over the choreic restlessness.

In Dunlap's laboratory at Wards Island, N. Y., I saw several cases, in which this author demonstrated the same changes.

The following case is also of interest. The clinical picture was that of choreiform disturbances lasting for nine years, when the hyperkinesis gradually subsided and was replaced by marked rigidity and definite dysarthria followed by contractures of all limbs. Anatomically, the striatum and pallidum showed a disappearance of all the small ganglion cells and a considerable involvement of the large cells. In a milder form, these same changes were also found in parts of the zona reticulata of the substantia nigra. We can conclude from this that the choreiform movement, disturbances, etc., are transformed into the akinetic-hypertonic syndrome of Parkinson as the degeneration of the striopallidum progresses, the large ganglion cells also becoming involved and the subpallidal centers joining the process.

In the cases manifesting psychic disturbances, the cortex participates in the degenerative process (Fig. 3). In most of these cases, the affection involves the frontal lobe and the centralis anterior, with the appearance of a peculiar glial pseudonuclear layer in the latter situated above the pyramidal or Betz cell zone. In the affected areas, there occurs a marked degeneration of the internal nuclear layer and the three lowest cortical layers.

This histologic picture is to be regarded as specific for chronic progressive chorea in connection with its characteristic localization.

The view that the locomotor disturbances in chronic progressive chorea are located in the striatum receives material support from the phenomena

of *toxic infectious chorea (Sydenham's chorea minor)* and of symptomatic chorea, dependent on various etiologic factors. In all these cases, there occur distinct, usually focal, alterations in the striatum. In these cases, however, unlike chronic progressive chorea, the small ganglion cell degeneration in the striatum is not as a rule emphasized to the same extent. A simple answer to the question of localization in these cases is, in view of the usually diffuse character of the manifestations, extremely difficult. The positive findings in the striatum in all such cases, however, lend considerable support to the theory presented herewith. This applies equally to toxic-infectious chorea, which develops with especial frequency after articular rheumatism and which is to be attributed partly to embolic and partly to infiltrative and degenerative focal processes, similar in character to the choreiform complications of epidemic encephalitis

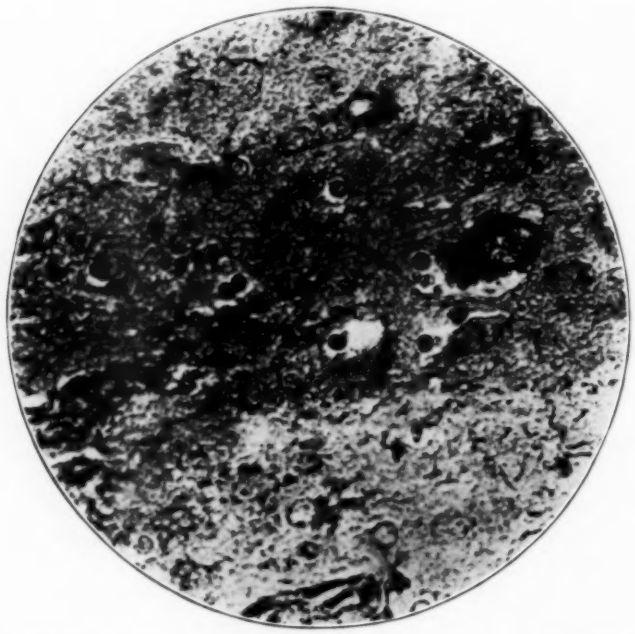

Fig. 4.—Spirochetes in a focus of dysmyelinization in the striatum from a case of general paralysis; Jahnel stain.

and the well-known changes accompanying this disease. A general diphtheritic infection may give rise to a symptomatic chorea, as was seen in a case examined by Globus in my laboratory; in this case the grave parenchymatous degeneration was limited to the striatum. Chorea gravidarum likewise depends on embolic lesions in the blood vessels of the striatum.

In *general paralysis* also the occasional choreiform movements that may occur during the course of the disease would indicate extremely marked alterations in the striatum (Fig. 4). We may also postulate that certain components of paralytic speech—and locomotor disturbances—may be referred to the usual alterations in the striatum.

This is also true of symptomatic chorea, in tumors and in cerebral diseases based on syphilitic or *arteriosclerotic blood vessel disease.* I have examined

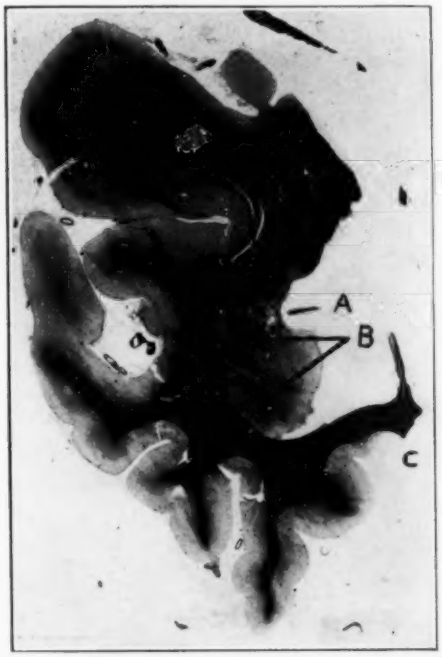

Fig. 5.—Apoplectiform facial tic in a case of cerebral arteriosclerosis. Only one focus was found in the pars anterior of the striatum. *A* indicates the focus; *B*, the striatum; *C*, the anterior commissure.

Fig. 6.—Paralysis agitans. Fatty degeneration of the striatum. Herxheimer stain.

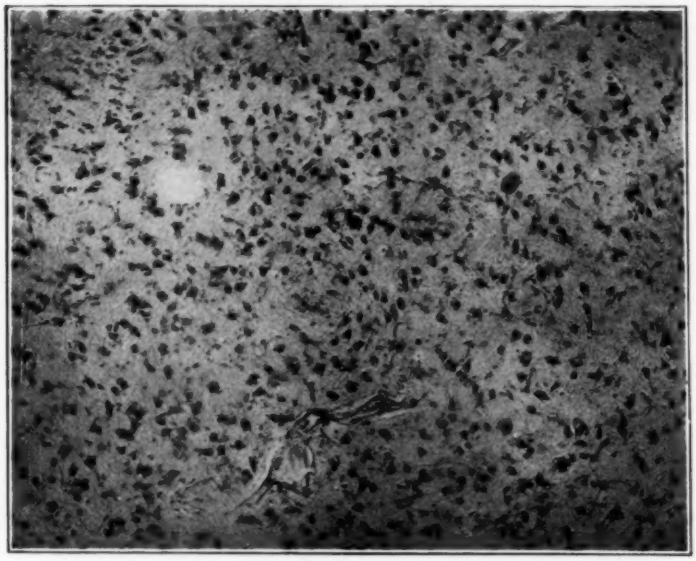

Fig. 7.—The striatum in paralysis agitans, showing the degeneration of the whole tissue, especially that of the large ganglion cells and relative decrease of these; the blood vessels show no marked changes. Nissl stain.

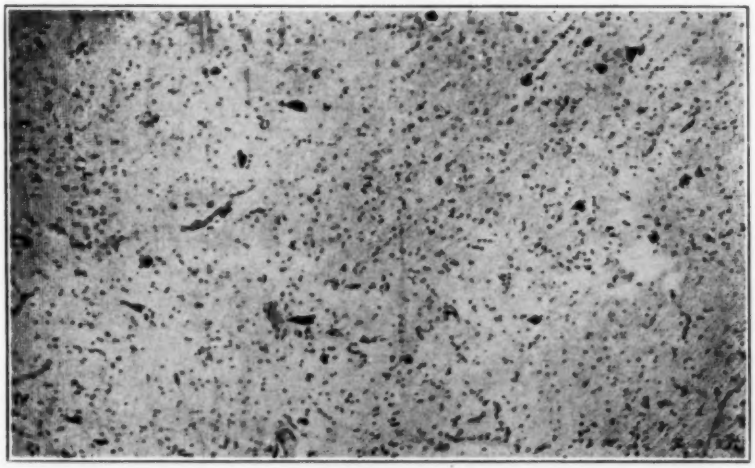

Fig. 8.—Pallidum in paralysis agitans. Degeneration and decrease in number of the ganglion cells. Nissl stain.

a patient with apoplectiform facial tic of one side and anatomically found an arteriosclerotic cyst in the capsule of the caudate nucleus of the opposite side (Fig. 5). Finally, such a condition may occur in multiple sclerosis and tuberous sclerosis when a corresponding area in the striatum is involved.

Senile chorea, an atypical form of a grave senile dementia, must also be placed in this group. There are cases in which the process of senile involution is markedly developed in the striatum as well as in the cortex, in which case the changes in the striatum represent in a certain sense a mild form of the disturbances that we encounter in genuine paralysis agitans. To illustrate, I can cite a case of senile dementia in which a tremor, subsequently developing

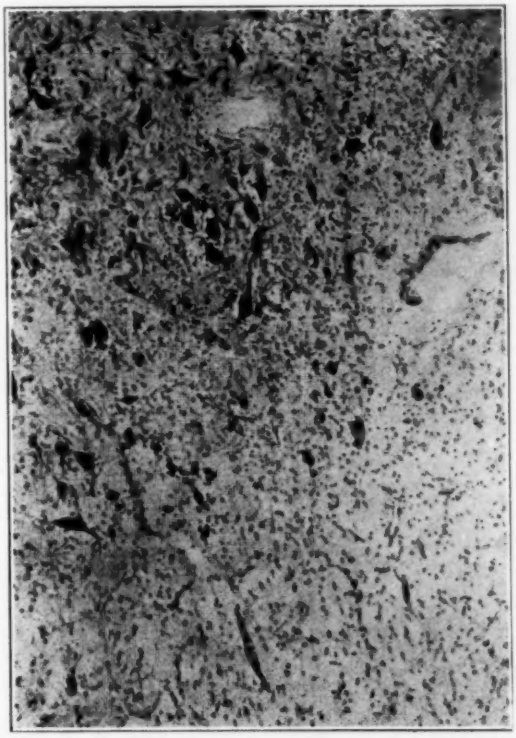

Fig. 9.—Substantia nigra in paralysis agitans. Foci of degeneration in the zona compacta. (This does not commonly occur.)

into a generalized chorea, was superimposed on a psychosis. The chorea was then followed by a marked rigidity of the lower limbs and a chorea at rest in the upper part of the body.

Anatomically, I found in this case the typical and marked senile changes in the striatum and pallidum, the pars anterior of the striatum showing a milder form of degeneration than the pars posterior. In the latter, all ganglion cells, the small and the large, were involved; some of them had disappeared and others showed marked degeneration. The clinically manifested chorea of the upper part of the body corresponded to the milder degeneration of the pars anterior striati, and the parkinsonism of the lower part to the severe

degeneration of the pars posterior striati. Senile chorea represents, to some extent, a connecting link between paralysis agitans and the locomotor disturbances of senility which are likewise localized in the striatum. The relation is emphasized by the fact that such senile choreas develop from a tremor and may terminate in partial or complete rigidity.

The clinical entities that are characterized by Parkinson's hypokinetic-hypertonic syndrome stand in sharp contrast to these varieties of chorea, so far as symptoms are concerned.

Parkinsonism is seen in its purest form in genuine *paralysis agitans;* it is anatomically dependent on a degenerative process which is akin to a process

Fig. 10.—Case of muscular rigidity caused by syphilis. There is a large focus (*foc.*) in the putamen; also secondary atrophy of the pallidum frontal section. *Caud.* indicates the caudatus nucleus; *l,* the lenticular nucleus; *caps. int.,* the internal capsule; *Nr.,* the nucleus ruber; *Put.,* the putamen; *Pall.,* the pallidum. Myelin fiber stain.

of senile involution, particularly with fatty degeneration of the neural parenchyma (Fig. 6) and degeneration of the large ganglion cells (Fig. 7). This process is, as a rule, located chiefly in the striatum and in milder forms in the pallidum (Fig. 8). In contrast to chorea, it is especially the large cells in the striatum that degenerate and are destroyed. In exceptional cases, the substantia nigra (zona compacta) may participate in the degenerative process (Fig. 9). The pathologic process may be accompanied by an intense capillary fibrosis and lesions resulting therefrom. In cases with unilateral predominance of the clinical symptoms, we have anatomically also a more severe damage of the striopallidum of the opposite side.

Closely akin to this clinically is Foerster's *arteriosclerotic muscle rigidity*, the lesions of which, determined by sclerotic vascular changes, are located in what apparently seems to be two favorite sites: At times there are definite vascular lesions in the fronto-ponto-cerebellar system; at others, in the striatum and pallidum and ultimately commingled with such processes in the substantia nigra.

Histologically, *senile muscle rigidity with dementia* must be carefully differentiated from these maladies (A. Jakob). This disease must be regarded as an atypical form of senile dementia, and represents, clinically and anatomically, a peculiarly grave end process of a typical senile cerebral process in the basal ganglions, especially in the striatum and in the pallidum. Corresponding to the clinical locomotor disturbances and in keeping with the gravity of the senile involutionary process in the striatum and in the pallidum (rarely also in the substantia nigra) in senile and presenile psychoses (and in Alzheimer's disease), there are found plastic transitions from the locomotor disturbances of the ordinary senium and those of Alzheimer's disease and senile chorea to senile muscular rigidity.

Similarly localized in the striatum and pallidum are those parkinsonian syndromes which not infrequently develop in syphilitic vascular changes, diseases resembling general paralysis, running a strikingly slow course and with variable serologic reactions at different times, mostly mildly positive or even negative (in blood and spinal fluid). In these cases, the vascular changes can be differentiated only with difficulty from typical arteriosclerosis, but usually the more intense vascular infiltration points to a syphilitic origin, as do the proliferative changes in the cells of the vessel walls—findings characteristic of endarteritis luetica of the small blood vesels. Congenital syphilis may give rise to such clinical pictures in childhood. We frequently find in these cases, in children as well as in adults, a bilateral necrosis of the striopallidum (Fig. 10).

At present, we frequently meet *parkinsonisms following epidermic encephalitis*. I have made most careful anatomic and histologic studies of five such cases, all of which showed almost identical symptoms, and have made the following observations with regard to localization: In the first case, which I published in 1921, the substantia nigra was greatly degenerated in the zona compacta; so was the pallidum, and, with diminishing intensity, the striatum, with conspicuous degeneration of the large ganglion cells. In two other cases, the intense degenerative process was limited essentially to the zona compacta of the substantia nigra. In the fourth case, there was in addition to the grave degeneration of the zona compacta of the substantia nigra an almost equally serious degeneration of the pallidum, and in the fifth case, the striopallidary degeneration exceeded the manifest, but receding, alterations in the substantia nigra. We see then that in individual cases of this disease the localization of the process may vary within certain limits but that the usual affection of the zona compacta of the substantia nigra stands in the foreground. These conclusions agree with the observations of K. Goldstein, the French school (Tretiakoff, Lhermitte and Cornil) and the American authors, all of whom emphasize the constant and almost invariably severe affection of the substantia nigra. More recent investigations in Russia and Italy point to similar changes in the striatum and pallidum, in addition to the disease of the substantia nigra, as in my first case. At any rate, we are justified in saying today that the *malady commonly spares the cerebral cortex and finds its chief location, as a rule, in the substantia nigra. From this it also follows that a degeneration*

of this gray area may condition a parkinsonism, as Brissaud probably thought as far back as 1895.

As I have already explained, on the basis of my first case, there is at the bottom of these cases a progressive and almost purely degenerative affection of certain gray centers, although on rare occasions it is possible to establish a mild lymphocytic infiltration of the blood vessels. But we may assume that the acute and subacute processes of epidemic encephalitis (which, as a rule, favor the substantia nigra) bring about changes in these gray nuclei, which practically recover from the inflammatory and infiltrative component but go on to a progressive nuclear degeneration.

But there is evidently a second and much rarer group of after-effects of epidemic encephalitis with a much more varied symptomatology and course; in such cases—often with a clinical picture of *pseudobulbar palsy*—we find a fairly extensive degenerative process (cortex and white matter, striatum, pallidum, thalamus) accompanied by a fat granular cell formation and more marked infiltrative vascular phenomena. The abortive type of metencephalitis chronica has been studied only clinically.

Finally, we have a parkinsonism representing the clinical picture of *Wilson's disease* and *Westphal-Strümpell's pseudosclerosis.* These, as we know, are diseases of adolescence with special emphasis on the constitutional component, in which there is almost always combined disease of definite central nerve nuclei, a peculiar cirrhotic change in the liver, and commonly extrapyramidal locomotor disturbances, together with more or less marked psychic changes. These two types of disease, between which there are plastic, clinical and anatomicohistologic transitions, offer us a pure degenerative process in the parenchyma, located in the striatum (and pallidum), dentatum and cortex. Wilson's disease, generally designated as "bilateral progressive lenticular degeneration," commonly presents a necrosis of the parenchyma combined with a formation of fat globular cells, whereas the atypical large glia cells of Alzheimer may be wanting. The pseudosclerotic process in this location, on the other hand, is commonly characterized by the occurrence of Alzheimer's large atypical glia cells, together with grave degenerative phenomena in the ganglion cells, but without a necrosis of the parenchyma, no fat granular cell formation and without formation of vacuoles and glia fibers.

The atypical large glia cells of Alzheimer, in my opinion and in keeping with my anatomic experience, do not show the evidence of embryonic appearance, but point to a peculiar acute glia reaction of a strong degenerative nature, produced by extraordinarily virulent toxins.

The etiology of both diseases and their relations to the affection of the liver and to the cerebral changes remain a mystery. The syphilitic etiology which was present in Homen's cases must by no means be generalized, for this was wanting in almost all the cases observed subsequently. Neither can we find any verification for the theory so much discussed lately that the hepatic disease brought about cerebral changes in the striopallidary centers owing to the absence of the detoxicating function of the liver. In my laboratory, we conducted careful experiments (Kirschbaum) both on human beings suffering from acute yellow atrophy of the liver and on experimental animals whose livers were damaged in various ways, but in no case could we find special affection of the basal ganglia, especially of the striopallidum.

The localization of the process in this disease is usually diffuse (striopallidum, dentatum and cortex cerebri), but we may say with certainty that the parkinsonism in these cases is the result of the striopallidal disease.

Besides the cases mentioned, there are many atypical cases in which it is difficult to determine to which group they belong. Three years ago, I described a case of a *catatonic psychosis with akinesis* of ten years' duration. The condition began between the ages of 30 and 40, and there was no familial background. Anatomically, I found the typical manifestations of Westphal-Strümpell's pseudosclerosis diffusely scattered. The cortex cerebri was the one mostly affected, then the dentatum and finally the striatum. In the white matter of the cerebrum and cerebellum, many foci with fat granular cells were present.

In the literature, we have three cases of *Wilson's disease* with a circumscribed lesion of the striopallidum — important in regard to the question of localization: The first case, described by Bielschowsky, showed, clinically, akinesis, with tremor, shaking and dysarthria, resulting in a marked rigidity. Anatomically, only a pure degeneration of the striatum was found. The other two cases had the same localization, i. e., the whole striatum and pallidum were somewhat affected, but the clinical pictures were different. The one case, described by von Economo, showed an outspoken rigidity with contractures, whereas the other, described by C. and O. Vogt, Bielschowsky and Thomalla, showed a torsion spasm. The anatomic difference in these cases was only a difference in the extent of the damage of the striatum; in the case of von Economo, the whole striatum was degenerated, and in the case of torsion spasm, the striatum was partly affected, and islands of normal structure were present.

We can conclude that purely diffuse degenerative processes of the striatum in the adult do not lead to athetosis, but to tremor, shaking and dysarthria. When the striatal degeneration increases, the akinetic hypertonia becomes manifest, with a possibility of the akinesis appearing sooner than the hypertonia. The torsion spasm is like the athetosis, based on a pallidal lesion, both being dependent on the relative functional capacity of the striatum. The striatum must be at least partially intact in order that the pallidal athetoid hyperkinesis may develop.

Henschen has also described, under *"spastische Pseudosclerosis"* peculiar types of cases in middle age, without clear etiology, with remissions, rapid progress and the following cardinal symptoms: beginning with anxieties, mild confusions and mild extrapyramidal symptoms, then gradually increasing in severity to a Korsakoff syndrome, with delirium, anxiety, confusion and optic and acoustic hallucinations. Besides these cortical symptoms, there are pyramidal tract symptoms, such as absence of abdominal reflexes, and occasional Babinski and Oppenheim signs but no marked paralysis. Of special note are the extrapyramidal symptoms in the form of tremor and shaking, increased tension in the extremities, akinesis, dysarthria, astasia-abasia and frequently marked pains. This condition terminates fatally in about one to three years, frequently with signs of irritability and paralysis of bulbar centers and marked mental deterioration. Macroscopically, a slight atrophy of the cerebral cortex and of the striatum is usually found. Histologically, the characteristic process is a progressive parenchymatous degeneration in certain areas of the gray matter. It consists of fatty degeneration of the ganglion and glia cells without fat granular cells. There is irregular ganglion cell degeneration, often with glia rosette formation; later, diffuse protoplasmatic glia proliferation and lighter glia-fiber production occur. In the severest cases, the condition terminates in a status spongiosus, with small microscopic vacuoles in the affected areas. Occasionally perivascular infiltrations are encountered.

The severest damage is seen in the cortex of the frontal lobes, in the centralis anterior, with more or less damage to the other cortical areas. The degeneration of the lamina granularis interna and of the three lower layers is especially apparent. Betz's pyramidal cells are completely destroyed. The striatum shows the same changes as the lower cortical layers, as do certain nuclei of the thalamus. Milder changes are found in the substantia nigra and pons and sometimes in the medulla oblongata or spinalis. Of the projection fibers, only the pyramidal tracts appear to be somewhat lighter stained.

The symptomatology may vary greatly; in the beginning, it may be mistaken for neurasthenia and multiple sclerosis, and in the terminal state it may simulate general paralysis or Alzheimer's disease.

The histologic process corresponds in many ways to metencephalitis. It is still an open question whether these histologically similar cases form a uniform etiologic-nosologic entity.

I have therefore put them into one group under "spastic pseudosclerosis" of uncertain etiology. Similar symptomatology is to be found in Wilson's disease and in cases with arteriosclerotic or syphilitic blood vessel degeneration. The differential diagnosis is given anatomically by the type of the histologic process and clinically by the absence of certain signs that indicate Wilson's disease or blood vessel changes.

In cases of cerebral arteriosclerosis we frequently see combinations of partial pyramidal tract and extrapyramidal lesions, even in relatively early senescence. Anatomically, we find multiple small arteriosclerotic foci in the gray or white matter of the centralis anterior, in addition to similar lesions in the striopallidum, thalamus or in the frontoparieto-cerebellar system.

THE ATHETOSES

The athetoses can be classified thus: (1) athetosis symptomatica (in cases of Wilson's disease, arteriosclerosis or syphilis cerebri, metencephalitis, etc.), (2) athetosis of early childhood and (3) athetosis of adolescence. I have demonstrated in the foregoing that torsion spasm is based on a focal pallidal lesion, and in the same sense also symptomatic athetosis has a pallidal localization. I can illustrate with the case of a patient with syphilitic muscular rigidity with an apoplectiform athetosis of the left arm. The striopallidum had small arteriosclerotic foci on both sides, but in the arm region of the right side, the pallidum showed a large lesion which had produced the athetosis of the left arm. Another patient with the same disease suddenly developed an athetosis of the lower extremities with a marked tendency to twisting movements in addition to the common signs of the arteriosclerotic muscle rigidity in the striopallidum; the pallidum, especially the ansa lenticularis, showed large foci in the pars posterior on both sides in the region for the lower extremity. The following case is of special interest and importance: A patient at first had arteriosclerotic muscle rigidity, then an apoplectiform conjugate deviation to the right, flaccid paralysis on the right side and in addition an athetosis in the left arm — of six days' duration. Anatomically, I found an evident status cribratus arterioscleroticus in the striopallidum of both sides, in addition to which there was a recent large hemorrhage in the striopallidum on the left and an old focus in the pars anterior pallidi of the right side. In my opinion, the athetosis was the result of the old focal pallidal lesion and the flaccid paralysis the result of the fresh lesion of the striopallidum. Thus we see that an old pallidal focus may be incited to an athetosis in the contralateral side by a fresh lesion in the striopallidum of the other side.

The innervation of the striatum and pallidum is mostly contralateral, but, to a lesser degree, also homolateral. This bilateral innervation explains how striopallidal damage of one side may be adjusted to a great extent, as well as the importance of special pallidal functional balance of both sides in the development of the extrapyramidal disturbances.

My findings show conclusively that *athetoid movements in the adult are found only in cases in which there are lesions in the pallidum.* If such apoplectiform lesions in the pallidum enlarge or multiply, however, the athetosis ceases, and rigidity sets in. *Like the striatum, the pallidum has a somatotopical localization corresponding to the various regions of the body:* The area representing the head lies orally, then follows that of the upper extremity, then that of the trunk and finally that of the lower extremity.

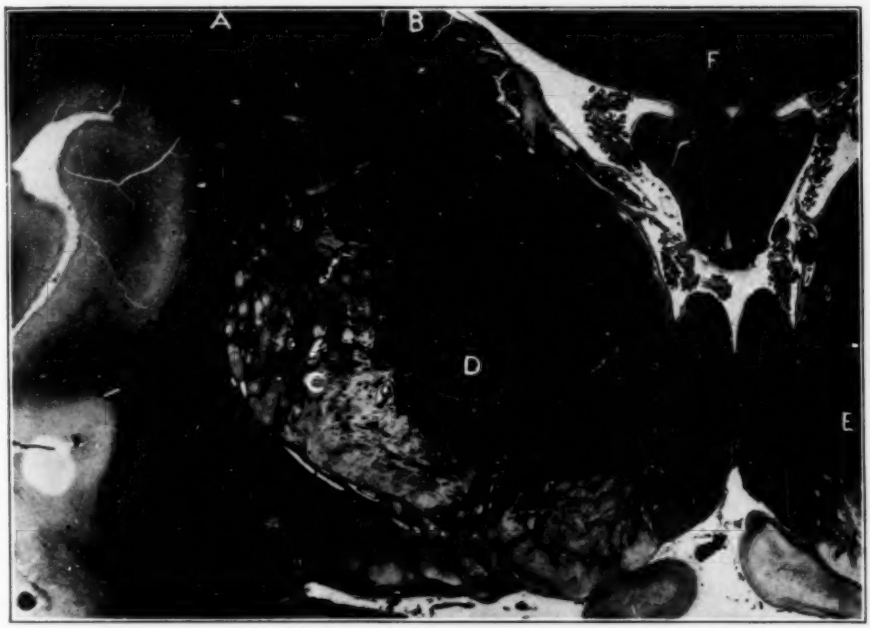

Fig. 11.—Status marmoratus of the striatum. Frontal section after a preparation by C. and O. Vogt. *A* indicates the internal capsule; *B*, the caudate nucleus; *C*, the putamen; *D*, the pallidum; *E*, the anterior commissure; *F*, the corpus callosum.

Next, we have to consider *athetosis occurring in earliest infancy* and forming a part of Little's disease (Fig. 11). This condition is congenital, and manifests itself in epileptic seizures, rigidity and athetosis, frequently showing a tendency to improvement. Psychic symptoms may be absent. Anatomically, we find the "status marmoratus" (C. and O. Vogt) of the striatum, which is a peculiar myelin fiber picture in the striatum and is the result of disturbance in development. It is the only affection of the striatum which produces pure athetosis.

Furthermore, we have athetosis in cases of infantile cerebral palsy. In such cases, we find the Bielschowsky type of cerebral hemiatrophy with a degeneration of the third layer of the centralis anterior, in addition to a

mild affection of the striatum, so that in these cases the athetosis was due to degeneration in both the striatum and laminae centralis anterior.

The question as to what constitutes, clinically and anatomically, the *infantile cerebral palsies* is as yet not settled. We can consider, however, as the main group that clinico-anatomic entity which is characterized in its syndrome by the typical triad of spastic hemiplegia, feeblemindedness and epilepsy, even though the causes are not always the same. Its anatomic characteristic features are as a rule unilateral cerebral defects along the distribution of the middle cerebral artery and the formation of external and internal cavities with marked distortion in the adjacent cortex.

This group of cases, of varying etiology, must be differentiated from other series of cases similar in symptomatology but clinically and anatomically different.

This series consists of:

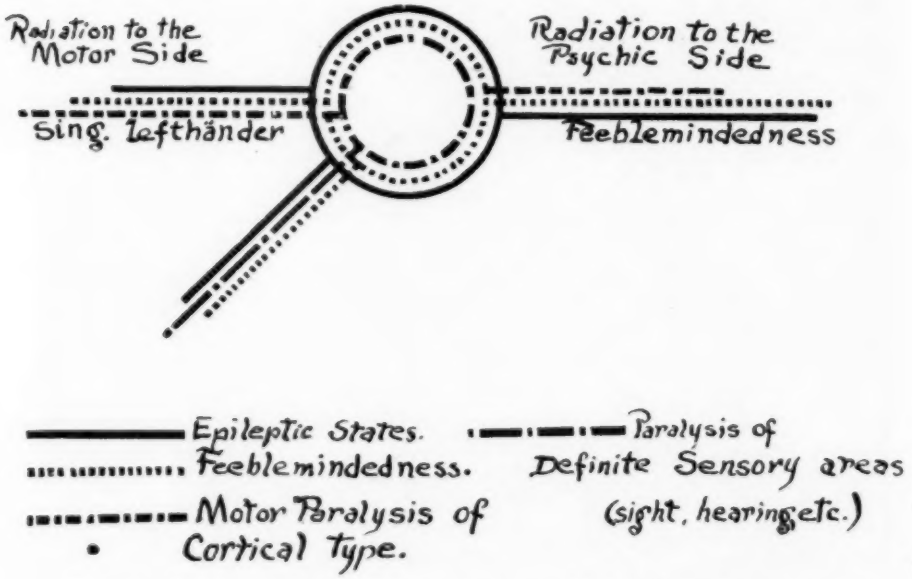

Fig. 12.—Scheme representing the main group of infantile cerebral palsy with its possible radiations.

(1) Malformations of the brain (macrogyria, microgyria, etc.), congenital hydrocephalus, tumors (especially plexus and ventricle tumors), syringobulbia, diffuse sclerosis of the white matter in childhood, and such etiologic or nosologic entities as multiple sclerosis in childhood, juvenile paralysis, tuberous sclerosis, as well as such familial diseases as Merzbacher-Pelizaeus disease or infantile and juvenile amaurotic idiocy.

(2) All outspoken striopallidal Little's disease, athetoses or rigidities. This group also includes the status marmoratus of the striatum, the status dysmyelinisatus of the pallidum of C. and O. Vogt (athetosis, terminating in rigidity), Little's rigidity due to congenital syphilis, infantile parkinsonism following epidemic encephalitis and in Wilson's disease, as well as the torsions-dystonia, double athetosis and infantile chorea occurring especially in pathologic processes affecting the basal ganglia.

In contradistinction to the last series, the group of infantile cerebral palsy first mentioned shows a characteristic pathologic process. The insult which affects the brain during normal intra-uterine or extra-uterine development brings about a lesion in the cerebrum, preferably in certain cortical areas (such as the area along the distribution of middle cerebral artery), resulting in a total or partial melting away of tissue, with subsequent scar formation. The otherwise normally constituted and developed hemisphere segments become involved in a more or less extensive cicatricial shrinking, whereby the vascularity and the exogenic etiologic factors are usually clearly emphasized, whereas the adjacent tissues become the seat of prominent secondary phenomena (such as extensive vacuole formation in the cortex and white matter, degeneration of certain layers in the cortex, etc.). The number of primary lesions, their distribution and the presence and size of the macroscopically visible vacuoles are, of course, important in determining the syndrome in individual cases, but for purposes of classification they are only of secondary importance. The anatomic factor common to all these cases is the scar formation in cerebral areas, mostly in the cortical zones, in relation to the blood vessel distribution. The main group of infantile cerebral palsy which is characterized by feeblemindedness, epilepsy and motor paralysis together with the corresponding lesions localized along the distribution of the middle cerebral artery, shows, at times, a tendency to disappearance of either the motor or psychic symptoms. So we see in some cases a gradual disappearance of the imbecility and the epilepsy, but retention of the motor symptoms (the "singular left-handed," stuttering); whereas in others, psychic symptoms (mental deficiency) remain, with a gradual disappearance of the motor paralyses and the epileptic attacks (paradoxic infantile cerebral palsy of Freud) (Fig. 12).

Clinically and anatomically considered, the group lends itself to the following classification:

A. Symptomatic Classification

- (*a*) Infantile cerebral palsy with motor paralyses
  - (1) Slight motor paralysis
  - (2) Motorspastic paralysis with athetosis (Bielschowsky's cerebral hemiatrophy with degeneration of the striatum) plus feeblemindedness and epilepsy
  - (3) Cases with grave spastic motor paralysis, feeblemindedness and epilepsy (the main and typical group)
- (*b*) Infantile cerebral palsy without outspoken motor paralyses (paradoxic forms)
  - (1) Epilepsy and feeblemindedness with indications of spastic manifestations, e. g., only a Babinski sign
  - (2) Feeblemindedness with epilepsy and ultimate speech disturbances
  - (3) Feeblemindedness with psychic epilepsy (epileptiform attacks of excitement)
  - (4) Cases with other lesions (blindness, deafness, ataxia, etc.), and corresponding lesions in silent regions of the brain.

B. Classification According to the Occurrence of the Insult and the Etiologic Factor

- (*a*) Intra-uterine
- (*b*) Traumas occurring during labor
- (*c*) Extra-uterine

In the last (*c*) especially, three important etiologic factors have to be considered:

(1) Strümpells' polio-encephalitis (probably related to the virus of anterior poliomyelitis).

(2) Various infectious diseases (scarlatina, typhus, syphilis, and other conditions).

(3) Trauma.

In addition to the foregoing, the *generalized bilateral athetoses* of childhood and adolescence have been present in all cases thus far described with disease of the pallidum (Fig. 13). The localization in most of these cases was of a diffuse character, but the pallidum was partly involved in all. This is also illustrated

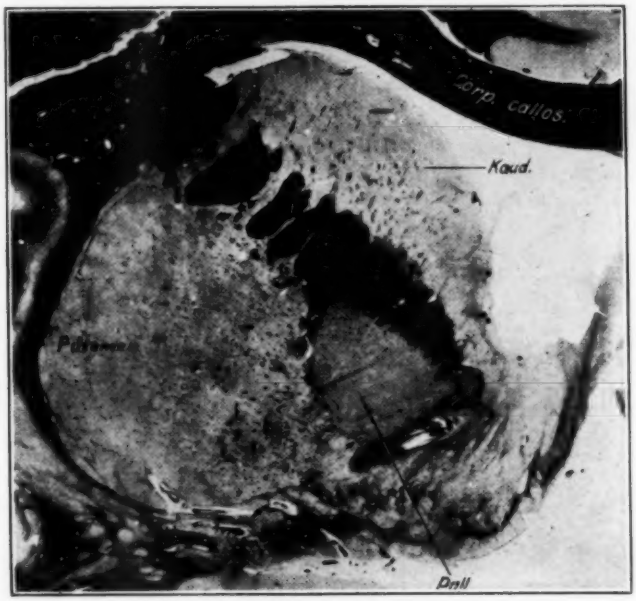

Fig. 13.—Double athetosis, frequently terminating in an akinetic hypertonic state. Frontal section showing the status dysmyelinisatus of C. and O. Vogt in the pallidum. *Corp. callos.* indicates the corpus callosum; *Caud.*, the caudate nucleus; *Pall.*, the pallidum.

by cases seen in Spiller's and Winkelman's laboratories. The slides were shown by these authors during my sojourn in Philadelphia. In this group should also be included the status dysmyelinisatus of the pallidum (C. and O. Vogt), with the clinical picture of athetosis terminating in rigidity, as well as the cases of O. Fischer and Rothmann, Richter, Hallervorden and Spatz, which showed peculiar changes in the pallidum. As further etiologic factors, the familial constitutional factor is emphasized in the cases of Hallervorden and Spatz; in other cases asphyxia at birth probably played a significant rôle.

We know that the pallidum has a special relationship (physicochemical) to certain poisons, especially carbon monoxid and manganese. In the cases of *carbon monoxid intoxication*, we find almost always a degeneration or necrosis of the pallidum. Of especial importance and interest in this respect is the case

described by Helene Deutsch, in which a bilateral necrosis of the nucleus lenticularis was found following an attempt at strangulation. Clinically, the case showed only a marked rigidity with contractures in the terminal state.

In a *unilateral lesion of the corpus Luysi,* there occurs a pronounced hemiballismus. I had a case of arteriosclerotic muscular rigidity following several apoplectiform insults. Two weeks prior to death, a left-sided flaccid paralysis appeared suddenly, followed the next day by a hemiballismus on the same side. Anatomically, I found the typical changes in the striopallidum on both sides accounting for the rigidity, and in addition a quite fresh lesion clearly and strictly localized in the right corpus Luysi. Similar cases are described by von Economo and O. Fischer.

A striopallidal parkinsonismus, when complicated by foci in the thalamus, especially in the lateral portion, will exhibit symptoms of flaccid paralysis with marked paresthesias, frequently astasia-abasia and eventually sleep disturbances. When the thalamic paralysis improves, the parkinsonian symptoms, together with a tremor, may return to some extent in the paralytic limbs.

At this point, I wish to cite a rare and interesting case, that of "a child without a telencephalon." It was born after a difficult and prolonged labor. Clinically, there was a marked and complete rigidity, and the child was capable of sucking, swallowing and faint crying; it lived only nine months. Anatomically, I found the whole cortex and myelin layer of the brain completely degenerated, only a part of the temporal lobe being fairly normal. The striatum was partly involved, whereas the whole pallidum, with the thalamus, cerebellum, midbrain and brain stem were normal.

Clinically and anatomically, this case was similar to the case described by Edinger and Fischer, in which the patient lived for three years. In both cases, there was marked rigidity of pallidal form in spite of the fact that the striatum was only slightly affected. This picture is one of decerebrate rigidity of Sherrington. The literature contains descriptions of similar cases of marked pallidal rigidity in infants and in adults in whom only both lenticular nuclei were affected.

Thus we see that in man the striopallidum is of the greatest importance in the development and coordination of movement in the infant as well as in the adult. This is contrary to experimental findings in animals, especially in animals without a telencephalon or thalamus. All these animals do not show decerebrate rigidity and can execute perfectly complicated movements, in spite of the absence of the whole cortex and of the striopallidum. Furthermore, they show normal tonus, can run, jump and climb, can change postures and can chew and swallow. The decerebrate rigidity appears only after injury to the red nucleus.

In man, however, these centers acquire great functional importance, so that the new-born infant depends entirely on a properly balanced activity of the neencephalon with the striopallidum, which assures undisturbed function for the red nucleus and brain stem. These experiences show the dangers of applying the criteria of animal experimentation to man in some cases.

These significant differences between animals and human beings may be due to various causes. Magnus believes that the changes in the red nucleus and brain stem in these conditions in man have not been sufficiently stressed. This does not coincide with my findings.

Of all the various explanations of this phenomenon, the following seems to me to be the most plausible.

We must assume that in human beings there has occurred an important displacement of functional activity in favor of our extrapyramidal system. Thus with the higher development of the cerebral cortex there has also been a greater functional differentiation in those areas, especially the striatum and dorsal thalamus, although they originated from the same matrix (alar plate of the prosencephalon). Even though the former, as C. and O. Vogt point out, including the pallidum, has not essentially undergone any higher anatomic development in comparison with the more highly developed monkeys, we must assume in man a higher functional differentiation, especially in the specific human functions (more highly developed gestures of reactions and expression, facial expression, speech, the upright gait, etc.). Special anatomic evidences are furnished us by the substantia nigra in human beings, in whom this structure has doubtless evolved more powerfully and with greater differentiation than in the highest mammals, and is characterized by the occurrence of ganglion cells containing melanin. As the latter shows this melanin first in the third or fourth year of life, the development of this gray nucleus offers a striking parallel to the physiologic differentiation of infantile movements in the acts of standing, walking and speaking. The facts of human pathology speak clearly for a similar displacement (change) in the functional significance of the other extrapyramidal centers.

A review of the individual clinical syndromes and their anatomic localization leads us to the following conclusions:

DISTURBANCES OF THE STRIATUM

Disturbances of the striatum may manifest themselves in the following clinical syndromes:

(1) Mild paresis of voluntary and involuntary movements; the synergic movements suffer most, while the isolated movements of individual members remain intact.

(2) Incoordination of synergic actions, especially sitting, standing, walking, speaking and all bulbar functions (striatal pseudobulbar palsy).

(3) Akinesias.

(4) Tremor and shaking; hypertonia and mobile rigidity.

(5) Absence of reflex abnormality, sensibility and psychic disturbances.

(6) In the adult, a striatal degeneration (especially of the small cells) constitutes the choreic syndrome with a mild form of akinesis and hypotonia.

(7) Milder affections of the striatum produce parakinesia, the severity depending on the extent of the damage.

(8) A slow destruction of the large ganglion cells of the striatum or of the whole striatum produces rigidity which in the diffuse processes is marked.

(9) Large apoplectiform lesions of the striatum produce contralateral flaccid paralysis which may frequently regress and lead to rigidity (the striatum, as stated, having bilateral action) more marked on the contralateral side.

(10) In the infantile as yet undeveloped brain, pure striatal lesions produce athetosis with hypertonia.

The pallidum syndrome consists of:

(1) Focal lesions—of athetosis and torsion spasm with rigidity (contralateral to the affected side) and often limited to the individual limb, according to the localization.

(2) Diffuse, but partial lesions of the pallidum produce general athetosis with a mobile rigidity. Diffuse complete lesions produce marked rigidity, followed by contractures.

(3) Absence of reflex anomalies, sensibility and psychic disturbances, and absence of vasomotor and temperature disturbances.

Lesions in the corpus Luysi produce hemiballismus. Lesions in the substantia nigra (especially in the zona compacta) produce rigidity of the parkinsonian type.

We must now turn to a brief discussion of an important question, namely, as to where *the real basis for all our extrapyramidal motor disturbances* is located.

We know that lesions in certain parts of the thalamus can give rise to choreiform movements, and we can therefore say that these disturbances are due to some change in the striopallidal functions brought about in an indirect fashion. But then disturbances of an extrapyramidal type are also found in cases in which there are lesions in the dentatum-rubral tract (Bonhöffer), and even in cases of Wernicke's poliencephalitis hemorrhagica superior. I had an opportunity to study such a case in my laboratory.

The rationale for locating all these motor disturbances in the midbrain and brain stem is furnished by a case described by Schilder in 1911. I would offer the following pathophysiologic explanation in this instance: The case was that of a man who for a long time had a spastic hemiparalysis in the right side and then suddenly developed marked athetosis on the paralytic side, which persisted for three weeks and terminated in death. Anatomically, Schilder found an old scar in the whole striopallidum and capsula interna of the left side, with secondary degeneration in the corpus Luysi and pes pedunculi and a recent focus in the right dentatum with a partial damage of this gray nucleus. The athetosis was the result of the focus in the dentatum, and the basis for the motor disturbance therefore could not be in the striopallidum which had been entirely degenerated for a long time, but must have been in the midbrain and brain stem. The midbrain and brain stem centers are subject to a double influence: In the first place, they are influenced by the efferent tracts of the cerebellum, and secondly, directly through the extrapyramidal system, especially by the fibers to the red nucleus and to the commissura posterior, whereby both pallida exert homolateral and contralateral influences. Thus numerous influences are brought to bear on the mechanism of the brain stem, whereby the maintenance of its functional balance is assured. If this balance should be disturbed at any point, incoordination of the brain stem function may develop. This produces a disruption of the extrapyramidally influenced automatism, the function of which is performed by these lower centers.

The harmonious cooperation of these systems assures the normal function of the brain stem mechanism, so that disturbances in function may be noted definitely only under certain conditions, whereas in other instances they are compensated for by the adjustment of other systems, and appear only in mild form or not at all.

When the whole striopallidum and dentatum of both sides are degenerated, the result is an extremely marked akinetic rigidity taking the form of Sherrington's decerebrate rigidity. I have had the opportunity to examine such a case.

If we are to utilize all these anatomic, physiologic and pathologic facts for the purpose of constructing a picture of the physiology of the extrapyramidal system, we can state the following:

We have in the dorsal part of the midbrain and brain stem an important motor coordinating apparatus, which as the result of being stimulated proprioceptively is the basis for all tone, station and posture. The red nucleus is the oral hypertrophy of this apparatus. In agreement with Magnus, we may consider the cooperation of the spinal reflexes, brain stem and red nucleus reflexes in the distribution of tonus in standing and in posture somewhat as follows:

The spinal cord centers for an extremity are reflexly stimulated through the posterior roots entering at the same level corresponding to the flexion and extension centers. To this level a tract descends from the centers in the posterior portion of the brain stem, which would bring about a decerebrate rigidity if it were the only agent acting on the spinal cord. This tract would, therefore, displace the tonus distribution in the spinal cord in favor of the extensor muscles. But, if together with this, impulses should come from the red nucleus through the rubrospinal tract to the same spinal cord segment, the tonus distribution would be displaced rather toward the flexor muscles. To a certain extent, the spinal cord centers are subject to two restraining forces: one having to do with extension and influenced by the medulla oblongata and one having to do with flexion and influenced by the red nucleus.

The resultant of these two influences is a uniform tonus distribution in the innervated organs. In addition there are fibers from the pyramidal tracts which end in the same segment of the spinal cord and carry chiefly impulses making for flexion, as experiments on cerebral stimulation show.

SYSTEMS INFLUENCING MOTOR APPARATUS OF MIDBRAIN AND BRAIN STEM

This motor apparatus of the midbrain and brain stem, especially the red nucleus, is influenced and stabilized by two systems:

(1) *The cerebellum.* The chief function of this proprioceptively stimulated organ is to intensify, to give position and to inhibit the whole coordinating apparatus, and more especially in the matter of controlling specifically those locomotor synergies which are determined by the direction of the movements or the falling of the body and its parts.

(2) Our *principal extrapyramidal system and its individual centers.* This system is built up as a whole on the coordinating mechanism of the brain stem and cerebellum, which it employs as a firm basis. The whole of this system, with the exception of the substantia nigra (which also derives direct cortical fibers), derives its stimuli from the thalamus and hypothalamus. The hypothalamic basis of the diencephalon seems to us to be of great significance in connection with the central regulations of all the metabolic processes of the organism, and more especially of muscle tone. The thalamus serves as a large reservoir afferently and efferently connected with the entire cortex, and receives proprioceptive and, especially, exteroceptive and cerebellar impulses, all of which bring to it information concerning the position of the body as well as the state of tonus of the total musculature and the deeper unconscious sensations. The thalamus is therefore an organ which not only orients the individual as to the changes in the state and tonus of his body, but also puts him in intimate relation with the outer world, this being of considerable significance for the sensations, feelings and emotions and, in a certain sense, also for the faculty of attention, for psychic activity and for consciousness as a whole.

Our extrapyramidal system — as the efferent organ of the thalamus and hypothalamus, in which the incoming stimuli are translated into highly developed

motor phenomena by virtue of the specific activity of these centers—thus appears to be a center for the movements of expression, the reactive flight and defence actions, the pain-protection and fright reflexes, automatic change of position and attitudes, assisting movements employed in locomotion, compulsive associated movements and the motor-partial components, which play a rôle in the actions of sitting, walking, standing, chewing, swallowing and speaking. It also exerts a regulating influence on the vegetative, hypothalamic centers which preside over the chemicophysical tonic processes, and is an organ for tone. It should be finally regarded as a part of the voluntary tract through which cortical impulses are directed on the anterior horn.

Hypokinetic and akinetic phenomena are to be attributed to a diminution of innervation due to the lack of stimulation from the extrapyramidal system and of discharges from its centers. The rigid components in the partial phenomena are based on the loss of the regulating function of the hypothalamic centers, the motor apparatus of the midbrain and brain stem and of the cerebellar (Sicherung—stabilizing) protection through the extrapyramidal system.

Chorea is to be regarded as an ataxia of pallidal functions conditioned by the striatum, especially by the omission of the small receptive and associative ganglion cell elements of the striatum. A tic is to be regarded similarly, but in this case circumscribed muscle areas in the striatum only are affected.

Complicated parakinesias are akin to choreiform phenomena and are to be attributed to a similar mechanism, but in these cases a milder damage to the striatum must be the main pathogenic factor. The more systemically the small cell degeneration in the striatum proceeds, the purer will be the choreiform character of the peculiar movements. Tremor and shaking seem to be, in part at least, determined by the striatum, being ataxic disturbances of coordination of the pallidary and subpallidary centers (perhaps also as peripheral mechanisms of muscle innervation) wherein the injury to the large and small striatum cells of the contralateral side is the important factor. Similarly, the relative injury of the striatum on the one side and of the pallidum on the other also plays an important rôle. Tremors and pulsions are also to be found almost exclusively in conditions of the substantia nigra, and are evidently expressions of tone disturbances.

Congenital athetoses of early childhood are to be attributed, in the corresponding diseases, to the abolition of the regulating function which the striatum exerts on the pallidum, caused by pathologic processes in the striatum. In such cases of athetosis, incoordinate and distorted mass movements similar to the mass movements of earliest childhood occur.

The athetoses of later childhood, early adolescence and adult life are to be regarded, together with torsion spasm, as ataxic disturbances of coordination having a pallidal origin (disturbance of pallidary function), in which the interferences with the static and kinetic coordinating mechanism in the midbrain and brain stem are particularly prominent. In contrast to the athetoses of adults, which depend on focal pallidary lesions, the generalized athetoses of later childhood and adolescence, corresponding to the diffuse pallidary degeneration, show a preponderance of subpallidary mass movements and mechanisms of the brain stem.

The chorea-athetotic phenomena which appear in connection with diseases of the region of the red nucleus, of the brachium conjunctivum (Bindearm) and of the tegmentum mesencephali (Hirnschenkelhaube) are ultimately to be referred to the brain stem and to be attributed to the release of the midbrain

(red nucleus) and brain stem from cerebellar control (Sicherung) on the one hand, and pallidary control (Sicherung) on the other.

It is important to distinguish these diseases from those that are produced by lesions located on the oral side of the red nucleus, where the hyperkinesias are to be regarded as indirectly caused by failure of striopallidal functioning in the sense of ataxic disturbances; the akinesias then seem to be flaccid paralyses of the striopallidum.

FUNCTIONS OF SINGLE CENTERS

On the basis of anatomic considerations and of lack of function in correctly localized pathologic processes, we may at present express the following views concerning the *functions of the single centers:*

The *striatum* is the true center for gestures of expression, reactive movements, etc. It also gives tone to the pallidum. The *pallidum,* which in the new-born is the center for the primitive and incoordinated automatic movements of earliest childhood, become in the adult the center for locomotor synergies of single muscle areas and portions of the extremities in the service of striate locomotor automatic acts, which are built on the locomotor synergies of the cerebellum and brain stem. In addition, it also exerts a significant influence in regulating tone.

The corpus Luysi apparently contrasts the locomotor synergies of whole sections of the body, with special emphasis on cerebellar balancing components.

The *substantia nigra* is to be regarded especially as a tone controlling center, which in a special way serves to determine the succession of movements.

The striatum and pallidum are somatotopically linked and, like the corpus Luysi, innervate both halves of the body, but with especial emphasis on the opposite half. Similar relations clearly apply also to the substantia nigra.

Finally, I should like to emphasize that *the whole extrapyramidal system is functioning when the thalamus is active, and as a result the pyramidal system has the most favorable condition for its own functioning, so that anterior horn cells are constantly receiving normally directing impulses through the working of the extrapyramidal system.*

News and Comments

EIGHTY-FIRST ANNUAL MEETING OF THE AMERICAN PSYCHIATRIC ASSOCIATION

The eighty-first annual meeting of the association will be held at the Hotel Jefferson, Richmond, Va., May 12, 13, 14 and 15, 1925.

ANNUAL MEETING OF THE AMERICAN NEUROLOGICAL ASSOCIATION

The fifty-first annual meeting of the American Neurological Association will be held in Washington, D. C., May 5, 6 and 7. The annual dinner will be held Wednesday evening, May 6, at the Army and Navy Club.

The annual golf tournament will take place at the Chevy Chase Country Club on Monday afternoon, May 4, the day before the regular meeting.

ANNUAL MEETING OF NATIONAL ASSOCIATION FOR STUDY OF EPILEPSY

The annual meeting of the National Association for the Study of Epilepsy will be held at Richmond, Va., May 11 and 12, 1925, immediately preceding the sessions of the American Psychiatric Association. Papers on the subject of epilepsy will be presented by Drs. Menninger, L. Pierce Clark, Damon, Gibbs, Tucker, Patterson and others.

DR. MELLA RECEIVES APPOINTMENT AT UNIVERSITY OF COLORADO

Dr. Hugo Mella, now acting head of the Department of Neuropathology at Harvard Medical School, has been appointed as Associate Professor of Neuropathology and Psychiatry in the University of Colorado Medical School, and Associate Director of the Colorado Psychopathic Hospital. He will begin his new duties May 15.

Abstracts from Current Literature

HYALINE DEGENERATION OF THE CORTEX IN GENERAL PARESIS. KONSTATIN LÖWENBERG, Ztschr. f. d. ges. Neurol. u. Psychiat. **93**:1 (Sept.) 1924.

Hyaline degeneration of the cortex is seldom described, but a few observations have been made. Billroth described the first case of this kind with a hyaline degeneration of the cortex. Alzheimer investigated two cases. In one paralytic patient, he found a significant volume increase in the basal ganglia, and in another he found focal areas in the cortex. He thought the hyaline substance was colloid. Mignot and Marchand described a brain of a paralytic person, a part of which showed amyloid degeneration; the vessels especially were affected in this case. The three latest contributions have been made by Sioli, Schröder and Dürsk. Sioli found a gelatinous swelling in a gyrus of a paralytic patient, which proved on histologic examination to be a collection of homogeneous substance in the brain tissue. Schröder described small concrements in the cortex which proved on chemical analysis to consist of calcium and an albuminoid substance. Dürck described a case of general paralysis which showed a number of oval homogeneous masses in the frontal lobes. Dürck thought the substance was colloid. Thus the cases reported in the literature demonstrate the deposit of a peculiar homogeneous substance in the cortex or more seldom in the basal ganglia. This substance is often connected with the vessels. Histochemically, in only one case was the substance found to be amyloid. Most of the older writers thought it was colloid, among them being Alzheimer, and, more recently, Dürck. Sioli speaks of an amyloid-like degeneration and Schröder of an albuminoid substance.

The author describes two cases of general paralysis with hyaline degeneration of the cortex. The first case was that of a paretic patient, aged 34, with a typical history. Postmortem examination showed that the dura had a fresh hemorrhage on its internal surface. The pia was thickened over the anterior two thirds of the convexity and over the base. The convolutions were atrophied. On section, the cortex was shrunken and the ventricles dilated. In the left temporal and parietal lobes, there was a gray-white appearance to the entire cortex, and nearby small foci of similar appearance were seen. The ependyma of the fourth ventricle showed granulations. Microscopic examination showed a typical picture except for the foregoing foci. The temporal lobe showed peculiar changes. The pia was thickened, lying in structureless lamellae. It formed a sort of coarse network in which were many types of cells — plasma cells, lymphocytes and polyblasts. The vessels showed a small, pale intima with shrunken cells, the media was structureless and thickened and formed a homogeneous ring. Only a few vessels showed a normal media, and even here the muscle fibers showed hyaline changes. Longitudinal section of these vessels showed a similar change of these throughout the course of the vessel. The cortex also was changed into a structureless, strongly refracting mass. The walls of its vessels showed hyaline degeneration just as in the pia. The parenchyma showed vessels thickly surrounded by mantles of plasma cells and lymphocytes, and in the parenchyma also was a huge overgrowth of neuroglia. The ganglion cells had all but disappeared, the remaining cells showing the picture of ischemic disease of Spielmeyer. Numerous astrocytes were found

in the parenchyma, and in some places a definite status spongivans was found. In most places, around the hyaline degenerated vessels, there were structureless hyaline masses in the surrounding parenchyma, giving a focal appearance to the entire process. Histochemically, the following was determined: with toluidin-blue the hyalin stained green-blue; with the van Gieson stain it stained light red to yellow-red; with hematoxylin and eosin, light blue; with Weigert stain, it stained blue black; with Mallory-Jakob stain, intense blue green. Other stains are also recorded. Similar areas of hyaline degeneration were found in the substantia nigra, in the red nucleus and in the pons. In these areas, the vessels and capillaries showed typical hyaline degeneration, and there was rich plasma cell infiltration. The focal nature of the disturbance was particularly evident in these places.

The second case is that of a woman, aged 59, who had general paralysis. The history and findings were typical of paresis, and she died eighteen months after admission to the hospital. Postmortem examination showed a thickened pia over the frontal lobes, more marked on the right than on the left, with numerous blue-black pigmentations. These were confined mostly to the pia, and involved the brain substance only in places. The cortex was definitely shrunken. Histologically, the pia was much thickened through the overgrowth of its connective tissue elements; lymphocytic and plasma cell infiltrations were abundant. The pia on the whole appeared hyaline in this case, and the vessels showed marked hyaline degeneration. In other details, it was similar to the first case. Besides the typical changes in the cortex, there were certain atypical changes in the vessels. The cells of the vessel walls were swollen, the nuclei dark and poorly demarcated from the cell bodies. In some places, they were grouped. A wall of lymphocytes surrounded the vessels, and miliary gummas were seen in the vessel walls. The perivascular gummas showed a typical structure. By a regressive metamorphosis, as the author puts it, the gummas in and outside the vessel walls assumed a hyaline appearance, and showed typical hyaline degeneration. The process is described as follows: The ground substance of the gumma assumes a homogeneous appearance; a mass of plump hyaline fibers with numerous lymphocytes therein takes the place of the ephithelioid cells, and finally a complete hyaline degeneration takes place. In isolated places, the larger arteries and veins in the cortex show the same process. The impression, as in the first place, is of a focal appearance of areas of hyaline degeneration in the pia and cortex, in and surrounding the vessels of these tissues. In neither case could amyloid be demonstrated, and the author uses the term hyaline degeneration to describe the condition of a homogeneous appearance in certain tissues.

As to the cause of the condition, the author suggests disturbance in nutrition as the most plausible explanation. In Case 2 especially, the condition is primary in the vessels and secondary in the parenchyma. But this is not always true, and one may assume that the process may be primary in the parenchyma, and is there taken up by the vessels. The author believes in his cases the changes in the parenchyma were secondary to changes in the vessels, and that this primary change in the vessels may be of different types. In the second case, it was a general hyaline degeneration of the blood vessels, and in the first case, a severe infiltration seemed to play an important rôle. Further explanation as to the causes of the degeneration, says the author, are not possible on the basis of his cases.

ALPERS, Philadelphia.

TABES (NEW STUDIES ON ITS PATHOGENY, PATHOLOGIC ANATOMY AND CLINICAL ASPECTS). G. R. LAFORA, Arch. de neurobiol. 4:97, 1924.

This paper is a review of recent work on the pathologic anatomy of tabes with a consideration of its early diagnosis and treatment. The findings of Schaffer and Richter are reviewed and their practical importance discussed. The importance of early diagnosis and treatment is emphasized, and clinical histories of subjects suffering from incipient tabes are given. These cases were correctly diagnosed by the author, but as the Wassermann test was negative, no ataxia was present, and as the patellar reflexes were present, the patients, on advice of their physicians, discontinued or did not follow the treatment prescribed, and their condition became worse.

CASE 1.—Tabes with persistence of patellar reflexes. The patient, aged 44, married, who had heredosyphilitic children, had contracted syphilis twenty years before, and had not received adequate treatment. The first children were syphilitic; the others were normal, owing to the antisyphilitic treatment received by his wife during pregnancy.

During the three years preceding examination, the patient complained of intense fulgurating pains in the legs, visceral pains and pains in the waist, rachialgia, slight disorders of micturition and constipation, diminution of potency and a sensation of hardening in the mammillary region. He consulted more than twenty physicians, among them several specialists, and was diagnosed as having arthritis or endocrinopathy, in spite of his calling their attention to the fact that he had syphilis. A negative Wassermann test and the persistence of patellar reflexes were regarded as a decisive argument against neurosyphilis.

The author found in examination: pupillary anisocoria, with preservation of reflex to light, exaggerated patellar and Achilles' reflexes; no Romberg sign (only hesitation) and no ataxia when walking. There was no ataxia in the hands. There were no somatic symptoms except an hypesthesic zone in the upper thoracic region. Analysis of the cerebrospinal fluid showed 27 lymphocytes per cubic millimeter. Globulin reactions (Nonne and Pandy) were positive; there was a slight Noguchi reaction. The Wassermann test was positive (++). The Sachs-Georgi test was positive. The Lange curve was 3345442000. A diagnosis of incipient tabes was made.

The patient consulted another specialist who assured him that the diagnosis was not justified since the patellar reflexes were still present, and that the only diagnosis that could be accepted was neurosyphilis.

CASE 2.—Tabes with unilateral diminution of the patellar reflex and bilateral diminution of the Achilles reflex.

The patient, aged 52, at the age of 22 had had a chancre which had not received adequate treatment. His wife had an abortion and no more pregnancies. For the last twelve years, he had had lancinating pains. He had improved somewhat during the last three years, but during this time he had sphincter disturbances, impotency, pains in the waist, progressive weakness and paresthesia in the legs, accompanied sometimes with painful cramps. There was no ataxia, although there was a slight hesitation in the Romberg sign. The left patellar reflex was almost completely absent, the right normal. The Achilles reflexes were absent. The pupillary reflexes were preserved. There was no hypotonus; superficial and deep sensibility were normal, but periosteal sense in the left leg was diminished. The patient could not stand as well on the left foot as on the other. Cerebrospinal fluid and blood tests were not made. The disease was diagnosed as incipient tabes.

The author emphasizes the fact that incipient tabes is usually monosymptomatic, as only a few spinal roots are affected. The absence of a positive Wassermann reaction in early and even in advanced cases of the disease is also a factor which adds to the difficulty of early diagnosis; yet the success of any treatment depends largely on the prevention of the diffusion of the lesions to healthy spinal roots.

From his experience, Lafora concludes that prolonged treatment is highly beneficial. In most of his patients, considerable improvement was noticed after treatment with small doses of bismuth and mercury preparations. Arsphenamin and silver arsphenamin when used at the beginning almost always resulted in increased painful symptoms; because of this, the author uses them only after a long treatment with bismuth and mercury.

Intraspinal injections of mercurial preparations associated with intravenous or intramuscular injections have always produced excellent and quick results, which far exceed those obtained by intravenous or intramuscular injections alone. Lafora begins treatment with a long administration of bismuth preparations. Injections of these substances into the cerebrospinal fluid carry the drug directly to the spinal roots affected. For mercurial injections, autogenous serum with doses not exceeding 2 mg. of corrosive sublimate is used, since it produces constant effects.

Several cases in which there was rapid improvement after a few intraspinal injections are mentioned. The effect of the treatment is soon seen in the relief of the gastric disturbances, which are not influenced by intravenous injections. In cases of intestinal disturbances, no improvement has been observed, although fulgurating pains and ataxic phenomena fast disappeared. With regard to the length of the treatment, Lafora agrees with other authors that since complete cure of tabes is doubtful, the treatment should be repeated two or three times every year throughout life. He has observed an arrest in the course of the disease in patients who have followed this advice.

NONIDEZ, New York.

ENDOCRINOLOGY AND OTOLARYNGOLOGY. EDITORIAL, J. A. M. A. **84**:371 (Jan. 31) 1925.

It is significant that the introductory contribution to the newly launched *Archives of Otolaryngology*[1] strives vigorously and frankly to liberate the devotees of the specialized practice in disorders of the ear, nose and throat from some of the shackles of pseudoscientific medicine with which they have all too often been encumbered. Readers of *The Journal of the American Medical Association* are aware of its attitude toward the various and numerous claims of endocrine therapy recorded in an enormous number of printed pages. Writing with the insight of the experienced investigator, Carlson[1] has clearly pointed out the tests which allegations of specific function on the part of endocrine organs must meet:

First, we remove the endocrine organs in question in otherwise healthy animals, and we note the natural history of the sequelae of such removal. Then we feed or inject this animal with various preparations of the organ removed, and determine to what extent these administrations change the natural history of the defects in the animal following the loss of the organ. Having such

1. Carlson, A. J.: Relation of the Endocrine Glands to Disorders of the Ear, Nose and Throat, Arch. Otolaryngol. **1**:1 (Jan.) 1925.

experimental data, we try to check these up on patients in whom we have evidence that the disease is due to hypofunction of a specific endocrine gland. By following the same rigid scientific control and reasoning as in the case of the experimental work, we can secure equally reliable data in the clinic as in the laboratory as to the efficiency of a particular endocrine product in a particular malady; but if we do not weigh the patient's statements when they are not checked up by objective tests, if we let our own enthusiasm run away with our judgment, if we forget the natural ups and downs in the malady, and, particularly, if we try a whole series of therapeutics simultaneously (irrespective of what the results in the patient might be), we can draw no reliable conclusions as to the specific effects of the organotherapy in question.

Considered from this standpoint, the gonads — the testes and ovaries — are at once eliminated from the competition for recognition; for they have not yet been accused of producing diseases of the ear, nose or throat. There is no reliable evidence that hyposuprarenalism, if such a condition actually exists clinically, can initiate disturbances of these organs. With respect to the pancreas, there is no indication that diabetic patients show an unusually high percentage of ear, nose and throat infections, or enlarged adenoids or tonsils, although they show some impairment of tissue repair in general and decreased resistance to infection. Carlson protests, in particular, against current attempts at roentgen-ray therapy of the hypophysis in certain types of deafness. It cannot stimulate, rejuvenate or restore tissues, as the blatant advertisements would lead us to believe. Careful experiments on the question whether the roentgen ray in any strength or any duration of application is capable of actually stimulating a gland have so far proved absolutely negative. Roentgen-ray treatment, in quantities that affect glands, seems to depress or destroy them without primary stimulation. At present, therefore, on the basis of demonstrated facts, the use of the roentgen ray to stimulate hypoactive glands is not only empiric but contrary to facts. There is no warrant for putting parathyroid tissue into the "pluriglandular pie" fed to patients. The parathyroids have been irradiated in cases of otosclerosis, despite the lack of evidence that hypofunction or hyperfunction of the parathyroids is a factor in the recognized diseases of the ear, nose and throat, even if we admit that the parathyroids are involved in bone and calcium metabolism. Carlson concludes that, as far as we can see at present, the thyroid is the only endocrine gland and the only field of organotherapy that calls for a serious survey and investigation from the point of view of possible relations to the infectious diseases of the ear, nose and throat, even in cases in which distinct cretinism and myxedema are absent. Those who are gullible may follow the faker's lead and deride such conservative, sane advice; but the profession at large should be thankful for an occasional critic who will pilot us safely through the "dense fog of pseudoscientific and pseudomedical writing."

PROGRESS IN THE STUDY OF CEREBRAL FUNCTIONS DURING THE PAST FIFTY YEARS. K. BONHOEFFER, Deutsch. med. Wchnschr. **50**:1708 (Dec. 5) 1924.

This number of the *Deutsche Medizinische Wochenschrift* marks the semicentennial of its existence. Several interesting reviews are presented by such men as Bonhoeffer, Wassermann, Romberg, Sahli and others.

Bonhoeffer reviews the progress which has taken place in the study of cerebral functions. According to him, the most outstanding figure of fifty years ago was Greisinger who emphasized the need of unity between psychiatry

and neurology, and because of this need, in 1869, he laid the foundation for the *Archives für Psychiatrie und Nervenkrankheiten*, and established a combined psychiatric and neurologic clinic. It was about this time that Erb presented his classical discussion regarding the pathology of peripheral nerve paralysis. This was soon followed by his work on tetany and other types of peripheral nerve involvement. It was Erb's stimulating work that led Heitzig and others to work on the cortex, and by their various experiments they soon found the principles of cerebral topography. Up to this time, the brain had been considered as an organ functioning as a whole. In 1874, Wernicke's work on sensory aphasia and its localization in the first temporal convolution was published. As might be expected, following this work a tremendous impetus to the study of cerebral localization was manifested.

The lines of development in neurology from 1860 apparently began with the periphery and worked centrally. Thus, first came the peripheral paralyses, atrophies; and in the late sixties, the spastic paralyses, tabes, and later multiple sclerosis.

Following Wernicke's work on aphasia, Liepmann took up the work of apraxia, and apparently demonstrated the dominating influence which the left cerebral hemisphere has over the right. The work of Marie and Monakow are discussed in passing. Monakow's chief contribution is the theory that the specific functions are not definitely isolated, but are correlated by association tracts and centers. The exhaustive work of Brodmann, who isolated over sixty different cortical areas, and Vogt, who isolated over 200 cortical fields, represents the extent and intensity of localization. As the author says, however, in spite of these advances, we still stand helpless before our problem.

The dogma that all psychic acts have their origin in the cerebrum has been laid open to serious doubts during the past twenty years. The work on chorea and muscle tonus, the work of Strümpell, Wilson and others on lenticular functions, and more recently the results of neuropathology in the field of encephalitis, have added much to our knowledge regarding the basal ganglia, the extrapyramidal system and the sympathetic system. That the subcortical areas have also a definite psychopathologic field can hardly be doubted in view of the motor and psychic disturbances noted in children following encephalitis.

During the past twenty years, with increasing knowledge regarding internal secretions, it has been found that all psychic processes are not confined entirely to the function of the brain, but, as is clearly shown by thyroid studies, disturbances of the intellect may occur as a result of their disorder. There is also a relationship between the internal secretion and the vegetative system. To enter on a discourse of this problem would be beyond the scope of the author's paper, and he simply wishes to call attention to the complicated mechanism which is at work correlating the central nervous system, the peripheral nervous system and the endocrine system.

The brain is an organ which contains the entire phylogenesis of human development, and therefore no other organ can approach it in importance. It is occasionally stated that if one assumes the theory of localization in the brain, one may run short of a place for the various functions. There is probably no need for alarm on this score. Gradually, as time goes on, added so-called neuroses are being isolated and placed on an organic basis. Endocrine disturbances are being classified, and at present "organneuroses" are being isolated and an attempt is being made to clarify the problem of psychogenic disorders.

MOERSCH, Rochester, Minn.

A Reflex Hallucinatory Process. J. M. Sacristán, Arch. de neurobiol. 4: 118, 1924.

The author presents a detailed study of a case of reflex hallucination in a patient who, owing to her culture, introspection, spontaneous tendency toward written auto-observation and approachableness, has greatly facilitated the analysis of the phenomena.

A nun, aged 32, had no known pathologic heredity; since adolescence, she had clearly shown the features of a paranoid constitution. After a sentimental conflict of homosexual nature with one of her girl students, a relation delirium, accompanied by depression and egotistic tendency, developed. This delirium later assumed the character of an intense excitation which caused her confinement in a sanitarium, where a series of pathologic alterations of perception were manifested, with absolute preservation of the affectiveness and intelligence. The process continued with varying intensity, finally reaching a minimum which allowed the return of the patient to normal life. The psychosis, regarded by Sacristán as a paranoid form of schizophrenia, still persists in the patient, who belongs to the pyknic type.

False optical perceptions began to appear with the character of pseudohallucinations, as described by Kadinsky. The patient described how she perceived the objects of the optical hallucinations in the form of a "luminous ribbon," and later she asserted that she could see two of the physicians of the sanitarium with the "eyes of her mind." False acoustic perceptions began to appear at the same time; these hallucinations were recorded by the patient during several months.

Bodily hallucinations were the predominant feature in the patient, giving her a subjective idea of sickness which the other symptoms of the psychosis could not give. At the beginning they were described as if "something were creeping along her body," arousing a "sensation of disgust." Later they were of a clear reflex character and were determined by real acoustic stimuli, such as closing of doors or words pronounced by certain persons. These hallucinations produced at the same time intense pain in the left side of the thorax or intense coldness and numbness in one half of the head. This type of hallucination was present throughout the course of the psychosis and always ended with sexual orgasm. The paranoid trend of her ideas during delirium was clearly defined in the type of her hallucinations which she considered as deliberately induced with the purpose of tormenting her, not so much with physical pain as with sexual sensations. These phenomena were produced only by the presence of certain women, seldom by men, a fact which the author interprets as due to homosexual constitution of the patient. The production of physical pain followed by sexual orgasm was regarded by the patient as a sign of possession by the persons who induced the hallucinations, and she expressed this idea by stating that "she belonged to them," that "she was their property."

Sacristán believes that the reflex hallucinatory process described belongs to the second group of the classification of Rorschach, and he discusses at length the main features of the case, as well as the mechanism of the phenomena involved. His interpretation of the case described is based on the fact that the patient was reprimanded for her fondness for the girl student already mentioned. This sentimental conflict started the psychic disturbance, since the patient thought that she was despised by everybody on account of her weakness. It seems likely that this affection of sexual nature, repressed by the patient, was later manifested in a somewhat different way, but always with a sexual character, being a repetition of the sensation experienced during her

relationship with the girl student, and now induced as a reflex through certain acoustic stimuli. This incident played the most important part in the clinical history of the patient, but final analysis of this aspect of the case was not possible.

NONIDEZ, New York.

CLINICAL OBSERVATIONS ON THE EFFECT OF PILOCARPIN IN NERVOUS DISEASES. J. J. RUSSETSKY, Ztschr. f. d. ges. Neurol. u. Psychiat. **93**:381 (Nov.) 1924.

This study was made in order to observe the effects of pilocarpin on the nervous system in healthy persons and in patients with nervous diseases. Fifteen normal persons were used as subjects, varying in age from 20 to 40. Fifty patients with nervous diseases were studied, thirty-three men and seventeen women. In most cases, the dose advocated by Petrén and Bauer, namely, 0.005 gm., was used, but in a few cases that of Eppinger and Hess was used, 0.01 gm. The following neurologic conditions were studied:

Hemiplegia	11	Amyotrophic lateral sclerosis	1
Paresis	2	Myelitis	1
Hemihyperkinesis rhythmica	1	Spondylitis	3
Hemiplegia alt	1	Laesio medulla spinalis	1
Epidemic encephalitis	8	Laesio caudal equinae	1
Tumor cerebri	1	Ischiatic neuritis	2
Chorea	1	Progressive muscular dystrophy	3
Acute cerebellar ataxia	1	Myasthenia gravis	1
Tabes dorsalis	3	Epilepsy	2
Syringomyelia	2	Hysteria	1
Lateral sclerosis	1	Neurasthenia	2

The effect of pilocarpin on the central nervous system may be summarized in the following table:

Effect After Injection of Pilocarpin	Beginning of Effect	Maximal Effect	End of Effect	Duration of Effect
Bradycardia	1 min.	10 min.	53 min.	52 min.
Slowing of respiration	3 min.	16 min.	29 min.	26 min.
Facial hyperemia	6 min.	7 min.	25 min.	19 min.
Drop in blood pressure	7 min.	27 min.	Over 1 hr.	Over 1 hr.
Salivary secretion	8 min.	16 min.	60 min.	52 min.
Secretion of sweat	10 min.	22 min.	65 min.	55 min.
Lachrymal secretion	18 min.	21 min.	31 min.	13 min.

In other words, the reaction of the circulatory system appears as the first and most lasting effect after injection of pilocarpin. The effect on the glands appears later and is less sustained. It affects, as shown in the table, the parasympathicotropic and sympathicotropic systems.

According to the author, the detailed study of the pilocarpin reaction is particularly significant in certain local diseases of the nervous system. It is possible to determine a definite status of the vegetative nervous system in these diseases. Such diseases are found in disturbances of the subcortical ganglia, the thalamus and in the medulla.

In general diseases of the nervous system, certain facts were noted. In hemiplegia, after injection of pilocarpin an increase in perspiration and vasomotor function on the paralyzed side occurred and a decrease in the secretion of perspiration on the healthy side. In encephalitis an increased salivary and lachrymal secretion, a marked hyperemia of the skin and, at the same time, a definite increase in perspiration occurred. In tabes dorsalis decrease or cessation of perspiration occurred after injection of pilocarpin. In progressive muscular atrophy, decrease in perspiration, marked myosis, salivary and lachrymal secretion occurred.

ALPERS, Philadelphia.

EXPERIMENTS ON THE TRANSPLANTATION OF PLACODES OF THE CRANIAL GANGLIA IN THE AMPHIBIAN EMBRYO. I. HETEROTROPIC TRANSPLANTATIONS OF THE OPHTHALMIC PLACODE UPON THE HEAD OF AMBLYSTOMA PUNCTATUM. L. S. STONE, J. Comp. Neurol. **38**:73 (Dec.) 1924.

Stone has previously published an experimental study of the development of cranial ganglia of *Amblystoma,* from which it appears that the cells of the adult ganglia are derived largely from the overlying cutaneous placodes. When the lateral line, epibranchial and general cutaneous placodes were removed before differentiation of neuroblasts had begun, the corresponding ganglia were absent. When only neural crest cells of the cranial region were removed, the cranial ganglia appeared to be normal, but deficiencies were produced in certain cartilages of the head. These experiments confirmed Landacre's conclusions reached independently from histologic studies that the neural crest of the head contributes to the formation of cartilages and other non-nervous structures. In the experiments here reported, Stone tests further the specificity of the cutaneous placodes for forming cranial ganglia by transplanting them to new positions.

The ganglion of the fifth cranial nerve is normally formed from two cutaneous placodes, an ophthalmic placode which contributes the cells of the ganglion of the maxillary-ophthalmic nerve and a gasserian placode which contributes the cells of the ganglion of the mandibular nerve. The ophthalmic placode can be transplanted on the same side of the head to a new position dorsal and posterior to the eye, where it forms an ophthalmic ganglion. This may be accomplished even though the graft is rotated 90 degrees in the anteroventral direction. When a right ophthalmic placode is grafted dorsal to the one of the host on the same side, it forms a ganglion which fuses completely or incompletely with the ophthalmic ganglion of the host. The extra ophthalmicus profundus fifth nerve which is formed may fuse entirely with the one of the host, or it may make with it a short common trunk or be entirely separated from it throughout. This appears to depend on the degree of fusion of the closely placed ophthalmic ganglia. When the ophthalmic placode of the right side is rotated 90 degrees and grafted posterior to the eye in place of the gasserian placode, it forms a ganglion which may or may not fuse with a partially regenerated gasserian ganglion of the host. The ophthalmicus profundus fifth nerve of the transplant in these cases may leave the fused gasserian and ophthalmic ganglion of the transplant singly or in common with the truncus mandibularis fifth nerve of the host. In either case, the two nerves anastomose at the angle of the jaw. Three cases suggest the conclusion that the ophthalmic placode can form a ganglion which entirely replaces the gasserian in form and function.

C. J. HERRICK, Chicago.

THE TREATMENT OF PATHOLOGIC URGES AND COMPULSION NEUROSES BY REEDUCATION BY GUIDANCE (ABLENKUNGSTHERAPIE). W. BECHTEREW, Ztschr. f. d. ges. Neurol. u. Psychiat. **94**:237, 1924.

Bechterew believes that there is something good to be said of all methods of psychotherapy, but that they must not be used indiscriminately and must be chosen with regard to the patient. Sometimes various or all methods must be used. A careful history of the case is essential. If the cause of the diseased state is not evident to the patient himself, this must be gone into most closely, and the starting point of the disorder, usually in early life, must be disclosed.

When this is laid bare, the patient should be told that his disorder is benign and completely curable. This certainly immediately improves his condition. The treatment begins at this point. To avoid distraction, the patient is placed in the reclining position and told to close his eyes and to concentrate on ideas of sleep, but not to go to sleep, paying strict attention to the physician. Thinking about sleep is necessary to eliminate all disturbing thoughts. The patient should remain absolutely motionless, not moving so much as a finger.

The author states that he then begins to talk to the patient, telling him that his illness is completely curable, and that as soon as he can free himself absolutely from all thoughts and connections with his compulsion, he will be contented and powerful. The patient is then told to concentrate on healthy ideas, and to substitute a new goal for the old, such as sports, in the case of onanism, and sweets in the case of excessive tobacco smoking. When he finds himself thinking about his compulsion he is advised to make a substitution.

In case of depressing thoughts, philosophic ideas should be instilled. Blushers and stammerers need encouragement, which is given by saying that people do not notice these things when talking, that all nervous patients blush readily and so on. Individual adaptation is the keynote. The seances should be continued for a period of time until the patient can get hold of himself. Sometimes two or three suffice, at times many more are without pronounced effect. Carried out in a passive, partial hypnotic state, this treatment is more effective than in the waking state. Finally, it is not enough to treat the psychologic condition of the patient, but his body also must be built up by a system of physical education.

FREEMAN, Washington.

MULTIPLE GLIOMA OF THE BRAIN AND CORD. H. W. MULLER, Schweiz. med. Wchnschr. **54**:1107 (Nov.) 1924.

In a review of the literature, Muller found only four cases of multiple gliomas of the central nervous system. To these, he adds a fifth; a man, aged 54, who in 1921 complained of a spastic, ataxic gait, bradylalia, and difficulty in speaking. At this time, it was noted that the deep reflexes were exaggerated, and the abdominal reflexes absent. A diagnosis of multiple sclerosis was made at this time. In August, 1923, the patient began to complain of severe headaches; Babinski phenomena, adiadokokinesis and astereognosis were present. By September, double choked disk was noted. Because of the increasing choked disk, headaches and ataxia, a diagnosis was made of a cerebellar tumor. The patient died, Sept. 19, 1923.

Postmortem examination revealed five isolated tumors: one in the cerebellum the size of a walnut, one in the third and fourth cervical segment about the size of a hazelnut, one in the second dorsal segment and two in the cauda equina. The remainder of the postmortem examination revealed nothing of importance beyond the finding of a lymphangioma in the gastrocolic ligament. Examination of the gliomas showed them to be markedly cellular, with a sparsity of fibers, and exceedingly vascular. The question of multiple sarcoma was considered, but at no place was there any indication of mesodermic involvement.

Metastatic gliomas are rare, and it is the author's opinion that one has to deal here with a system disease of the glial structure of the central nervous system. Muller believes that there is a congenital disposition of this system to tumor formation, and his belief in this congenital disposition is strengthened by the presence of the lymphangioma in the gastrocolic ligament. The author

excuses the diagnosis of multiple sclerosis on the grounds that the multiple tumors presented such a complicated picture as to indicate a multiple lesion picture.

MOERSCH, Rochester, Minn.

PROGRESSIVE LENTICULAR DEGENERATION. J. G. GREENFIELD, F. J. POYNTON and F. M. R. WALSHE, Quart. J. Med. **17**:385 (July) 1924.

These authors discuss seventy clinical cases, and thirty cases with pathologic studies from the literature, and report the case of a previously healthy girl of 14 years who, following a trivial injury to one foot, gradually developed an inability to keep her mouth closed, had drooling of saliva and dysarthria. One year from the onset, she had a masklike face, and showed spasmodic weeping and laughter. Saliva trickled from the half open mouth. There were limited range and rate of movements of the facial muscles and tongue, and the arms and hands were held in an attitude of flexion. During the last eight months of her life, she had become completely anarthric, had developed dysphagia and an increased frequency and intensity of the spasmodic weeping and laughing. She had rigidity of the facial and upper extremity muscles, a general flexion attitude of the trunk and limbs, tremor of the hands, and finally, rigidity with slow flexion-extension movements of the lower limbs. The periphery of the cornea showed a zone of greenish haziness. Death occurred twenty months after the onset. At necropsy (twelve hours postmortem), the liver showed marked atrophic multilobular cirrhosis. Gross examination of the brain showed marked shrinkage of the corpus striatum, involving the caudate nuclei, without changes in the globus pallidus. Microscopically, there was degeneration of the nerve cells in the putameni and caudate nuclei, with neuroglial overgrowth. There were similar though less marked changes in the globus pallidus and nucleus ruber. With Marchi preparations, the tracts leading from the putamen to the red nucleus and corpus Luysi, the posterior longitudinal bundle and the superior cerebellar peduncles showed degeneration. These authors prefer the term hepatolenticular degeneration, and state their belief that the cases described by the German writers as pseudosclerosis belong in this group.

POTTER, Akron, Ohio.

CONCERNING THE TREATMENT OF EPILEPSY BY MEANS OF THE PASTEUR VACCINE AND THE INJECTION OF STERILIZED MILK. N. D. OSSOKIN and S. M. OCHSENHÄNDLER, Schweiz. Arch. f. Neurol. u. Psychiat. **15**:60, 1924.

Protein therapy in the treatment of epilepsy is nothing new. Darkschewitsch gave subcutaneous injections of brain extract with the idea of increasing the immunity of brain tissue. The treatment of epilepsy by means of rattlesnake poisoning was conducted along similar lines. In 1914, Nikitin treated epilepsy with a Pasteur vaccine and submitted favorable reports. Bondarief and Worobjowa utilized sterilized milk. The authors were led to try this treatment because of the difficulty of obtaining drugs during the war. The vaccine, as a 1 per cent. emulsion of a cord dried three days, was given under the skin of the back. In adults, 0.5 to 1 to 2 c.c. were given daily. The treatment was continued for thirty days. After interruption of a week or several months, the second course consisting of thirty injections was given. Sometimes a third course followed.

In 25 per cent. of cases, considerable improvement resulted; in 50 per cent., the result was merely satisfactory; in 25 per cent., it was entirely ineffective.

This treatment was sometimes combined with the administration of chlorotone, which enhanced the favorable result. In must be added that chlorotone used alone was not followed by such favorable improvement.

Sterilized milk was given in twenty-three cases which were under observation for from five months to one year. Milk from the same cow was filtered, sterilized for twenty minutes, and injected every three to four days. In 17.4 per cent., a good result was obtained; in 43.5 per cent., a relatively good result was noted. On the whole, the Pasteur vaccine was followed by a larger incidence of improvement. Psychic equivalents remained entirely uninfluenced.

WOLTMAN, Rochester, Minn.

METASTATIC BRAIN TUMORS. TOULOUSE, MARCHAND and PEZÉ, L'Encephale **19**:414 (May) 1924.

When there are no symptoms indicating focal destruction or intracranial hypertension, a brain tumor may produce a mental state which cannot be differentiated from dementia due to other causes. In such cases, the history of the patient is exceedingly important.

The authors report in detail the case of a woman who, two months before her death, showed confusion and amnesia. When admitted to the hospital, ten days before her death, she was completely disoriented, and was incapable of any intellectual effort. She complained of fatigue and pain in the head. She was somewhat depressed and agitated, but there were no delusions or hallucinations. Physical and laboratory examinations revealed no visceral disease. There were no objective evidences of focal disease of the central nervous system, and the fundi were normal. Three years before her mental symptoms appeared, the patient had been operated on for cancer of the breast. There were no evidences of local recurrence.

Necropsy revealed many tumors in the left cerebral hemisphere, varying in size up to about 3 cm. in diameter, and in the right hemisphere one tumor the size of a pigeon egg and one the size of a large hazelnut.

Microscopic examination of brain tissue revealed evidence of inflammation near the metastatic growths.

The authors believe that the mental symptoms were the result of a meningeal reaction and of the inflammatory changes occurring within the region of the metastases.

HYSLOP, New York.

OBSERVATIONS ON THE NATURE OF THE MUSCULAR RIGIDITY OF PARALYSIS AGITANS AND ITS RELATIONSHIP TO TREMOR. F. M. R. WALSHE, Brain **47**:159 (May) 1924.

In his experiments, Walshe injected from 8 to 25 c.c. of sterile 1 per cent. procain solution into the motor points of the biceps, triceps and forearm muscles and studied the results of the deafferentation in paralysis agitans and postencephalitic Parkinson's syndrome. The author summarizes his observations as follow: Intramuscular injection or 1 per cent. solution of procain abolishes parkinsonian rigidity but leaves voluntary power of the muscles unimpaired. The rigidity, if not the sole factor in the causation of the characteristic slowness of initiation and restriction of range of voluntary movements in paralysis agitans, is a potent factor, since movements employing muscles rendered flaccid by procain injections are notably more rapid and ample than the same movements carried out by the rigid muscles before the injections. The tremor is not affected by the protein injections and therefore

must be essentially different in origin and nature from the rigidity; it is extremely probable that the effect of the procain is due to a selective paralyzing action of the drug on the afferent nerve fibers, hence the atonic muscle is the result of deafferentation, and parkinsonian rigidity must be regarded as a true proprioceptive reflex action. It is possible that the abolition of the rigidity is due in part to a local effect of the procain on the muscle fibers. More knowledge as to the influence of posterior root section on this form of muscular rigidity is required before these points are finally settled.

POTTER, Akron, Ohio.

RELAPSING FEVER THERAPY OF NEUROSYPHILIS. F. PLAUT and G. STEINER, Ztschr. f. d. ges. Neurol. u. Psychiat. **94**:153, 1924.

Plaut and Steiner make a further report on their results in the treatment of neurosyphilis by means of the spirochete of relapsing fever. *Spirochaeta obermeierii* is inoculated subcutaneously from the blood of a mouse infected with the disease. The course of the disease is benign, lasting for a variable length of time and then subsiding spontaneously. Transmission of infection is practically a negligible danger, and those who have had the disease are resistant to future inoculations. The treatment may be carried out by the physician in private practice. Complications are seldom seen and are usually benign. The authors believe that it will take a long time to evaluate the results, perhaps ten years, but in the present communication they deal with the results in eighty-three patients treated between 1919 and 1922. Of these, eighteen are dead, thirty-three have been discharged and twenty-five are still in the institution; 34.5 per cent. had very good remissions.

Patients with late cases were not benefited; if anything, their end was hastened, and they gave little reaction to the infective treatment. A high lymphocytosis of the spinal fluid was considered a good prognostic sign. In refractory cases, both relapsing fever and malarial treatment were tried. The Wassermann reaction on the blood became negative in about a third of the patients treated. The spinal fluid proved more resistant, though the cytology often became normal after treatment.

FREEMAN, Washington.

CHANGES IN VASCULARITY IN THE BRAIN STEM AND CEREBELLUM OF THE ALBINO RAT BETWEEN BIRTH AND MATURITY. EDWARD HORNE CRAIGIE, J. Comp. Neurol. **38**:27 (Dec.) 1924.

The author has previously published quantitative studies of the vascularity of different parts of the brain of the adult rat, and here presents data on the development of the vascular pattern. The capillary and vascular density is less at birth than at maturity, and the relative vascularity of the various centers is not the same as in the adult. There is a slight increase in vascularity in some parts during the first five days of life, and there is a slight decrease in others; but practically all parts studied, except the cerebellar cortex, show a definite increase during the second five days when motor activity is developing. Between the tenth and twenty-first days, there is rapid increase in vascular richness in all the regions studied, and the differences in relative vascularity between the various centers which are characteristic of the adult become established. Vascular richness is related to functional activity, and this sort of activity requires a greater blood supply than do the processes of growth.

C. J. HERRICK, Chicago.

BLOOD FERMENTS IN EPIDEMIC ENCEPHALITIS. ALPERN and LEITES, Ztschr. f. d. ges. Neurol. u. Psychiat. **94**:35, 1924.

The authors in an investigation of forty cases demonstrated that blood protease is increased in the influenza-like, somnolent and myoclonic types of the acute disease and in progressive forms; it varies within normal limits in the parkinsonian forms. Blood catalase remains normal or is slightly diminished in the influenza-like and somnolent forms, while it is elevated in the parkinsonian and myoclonic forms.

FREEMAN, Washington.

MESENCEPHALITIS SYPHILITICA. S. A. K. WILSON and S. COBB, J. Neurol. & Psychopath. **5**:44 (May) 1924.

This paper presents a summary, from the literature, of twenty-two cases of tabes or general paresis with paralysis agitans and a detailed report of four personally observed cases of tabes with associated Parkinson's syndrome. The authors believe that the Argyll Robertson pupil and the tremors of paralysis agitans are not merely coincidental, as held by most of the former investigators, but are due to syphilis of the midbrain, i. e., lesions in the vicinity of the red nucleus, substantia nigra, corpus striatum, or in the afferent or efferent tracts passing through the mesencephalon or the subthalmic region.

POTTER, Akron.

REPORT OF A FATAL CASE OF DIABETIC COMA WITH INSIGNIFICANT KETONURIA, AND WITH A LARGE AMOUNT OF ACETONE IN THE SPINAL FLUID. HENRY M. FEINBLATT, Arch. Int. Med. **34**:508 (Oct.) 1924.

In the unusual case of diabetes reported here, the disease ran its course in three weeks. The patient was a girl, aged 9. Profound coma developed about three days before death. In spite of treatment by lavage, sodium bicarbonate, and administration of 110 units of insulin within twelve hours, the patient showed no response to the medication. As indicated by the title, the striking feature of the case was the absence of acetone in the blood, the presence of only a trace of acetone, and no diacetic acid, in the urine, while the spinal fluid gave evidence of a very large amount of acetone.

VONDERAHE, Cincinnati.

THE VENTRICULUS TERMINALIS: ITS GROWTH AND DEVELOPMENT. J. W. KERNOHAN, J. Comp. Neurol. **38**:107 (Dec.) 1924.

The dilated central canal at the lower end of the human spinal cord is present in all fetuses of more than 22 mm. in length, and is found in the conus medullaris and filum terminale of all children and adults examined. This is a true ventricle without communication with the subarachnoid space. It attains its maximum size at about two years after birth, and is of irregular and variable shape.

C. J. HERRICK, Chicago.

Society Transactions

PHILADELPHIA PSYCHIATRIC SOCIETY

Regular Meeting, Nov. 14, 1924

EARL D. BOND, M.D., *President, in the Chair*

PSYCHOSES IN TWINS. DR. ALFRED GORDON.

Attention was called to the cases in literature in which a mental affection occurred in one twin immediately after it had occurred in the other, or much later, or after the recovery of one of them, or simultaneously in both. Finally, it is stated that mental disease may occur in twins who have been separated from one another for years. The author presents the histories of two pairs of twins in each of whom the psychosis ran an identical course in character, duration and termination. The conclusion reached from all the cases reported and his own is that a psychosis especially characteristic of twins cannot be admitted. The only inference that can be drawn is that if the predisposition to mental disorders in many instances presupposes a congenital morbid organization of the nervous system, the rôle of heredity finds its strongest corroboration in cases of psychoses in twins. All such cases point most emphatically to the fact that similar physical organizations of the nervous system may lead to similar pathologic disorders. The occurrence of a parallelism in morphologic and pathologic elements in twins in the cerebral organization and in its physiologic functioning speaks strongly in favor of single ovum twins, especially when both are of the same sex. Psychoses in twins develop frequently on the basis of degenerative etiologic factors, and the similarity of the mental disorder finds its raison d'etre in a consanguinity which surpasses the ordinary limits.

A CASE OF GASTRIC CARCINOMA SIMULATING INVOLUTIONAL DEPRESSION WITH SOMATIC DELUSIONS. DR. CLIFFORD B. FARR.

The patient was an unmarried woman, aged 48, who gave a history of mild depression sixteen years before. The present attack was of one year's duration following pain in the teeth and sore gums. She developed dyspeptic symptoms and vomiting; food stuck in her throat. She could take nothing but liquids for six months. She said that she had not had fruit or vegetable juices during this time. Her teeth were extracted five months ago. She had lost 60 pounds (27.2 kg.) in weight. She was mentally depressed, agitated and insisted that she could not swallow, that there was no room for food. "I am depressed but it is all physical; nothing nervous and no mental trouble." On admission to the Pennsylvania Hospital for Mental Diseases, she was exhausted and febrile; her gums were boggy; there was extensive purpura; her abdomen was distended, and the spleen was enlarged. Rectal feeding was necessary as a duodenal tube could not be passed. Necropsy showed: verrucose endocarditis of the aortic valves, splenic and renal infarcts but no organisms in the heart blood. There was an infiltrating carcinoma involving the whole stomach except the pylorus, and producing obstruction at the cardia. There was involvement of other structures by continguity. Three cases were cited in which delusions were associated with organic lesions such as perforated

duodenal ulcer, cancer of the colon, etc. Attention was also called to cases of stomach and duodenal ulcer seen at necropsy in mental cases without corresponding symptoms.

DISCUSSION

DR. J. HENDRIE LLOYD: This case reminds me of a case of locomotor ataxia at the Philadelphia Hospital not very long ago. The patient became extremely anemic and had all the blood appearance of pernicious anemia. Although he gave a history of syphilis and there was an Argyll Robertson pupil, we felt that possibly it was a case of cord disease caused by pernicious anemia. Necropsy showed cancerous disease of the pyloric end of the stomach. The patient had no symptoms referable to his stomach condition, such as pain, nausea and vomiting. This was accounted for by the fact that he did not have consciousness of pain owing to the tabetic analgesia. Absence of pain is seen in other tabetic lesions such as the arthropathies and perforating ulcer.

DR. CHARLES W. BURR: There was an insane negro at Blockley some years ago, who had the delusion of a living animal in his stomach. He was ignorant, having been brought up in Louisiana, so the mere fact that he believed that something lived in his stomach did not prove that he was insane. He was an insane man for other reasons. When his body came to necropsy, there was a polypus attached by a pedicle to the wall of his stomach. The pedicle was some 2 or 3 inches (5 cm.) long, and movable, and I believe that the man had sensibility enough in his stomach to feel some foreign body there which he interpreted in this delusional way. The patient was thought to be a chronic alcoholic.

DR. CLIFFORD B. FARR: A striking point about this particular case was that the patient used the same phraseology that depressed patients habitually employ. "Food would not go down," "It stuck in her throat." She insisted from the first that she had cancer of the stomach. She was justified in every respect; there was no delusion at all. I saw Dr. Lloyd's patient at the Philadelphia Hospital, but did not think it necessary to make use of the diagnosis of locomotor ataxia to explain the lack of pain. It is not uncommon to find carcinoma of the stomach without pain so long as the orifices are not involved.

THE MENTAL DISORDERS OF CHILDHOOD. DR. CHARLES W. BURR.

Dr. Burr discussed the importance of studying mental peculiarities in children, as well as definite insanities, in order to foretell a child's future. Acute mania is mentioned as the one typical insanity occurring in young children. Eight cases of juvenile paresis were reported. All of the patients had congenital syphilis. The rarity of two children in the same family suffering from paresis was mentioned. On the other hand, in this series of cases practically all the brothers and sisters had some form of chronic illness affecting the central nervous system. Cases of personality changes following epidemic encephalitis were referred to in which children who had previously been normal became vicious, lying, cowardly and often criminal without marked signs of organic nervous disorder.

DISCUSSION

DR. J. HENDRIE LLOYD: I am particularly interested in what Dr. Burr has said about juvenile paresis. He has given an interesting collection of cases. It would be particularly interesting to know whether careful studies have been made as to paresis in the parents. The problem is practically the same as in

conjugal paresis; that is to say, whether or not there is a particular strain of the spirochete that causes paresis or locomotor ataxia. We know that there are a number of observations which raise the presumption in favor of this, and in these cases of hereditary paresis we might gather valuable data, since two or three of Dr. Burr's cases showed that the parents had paresis.

DR. FRANCIS X. DERCUM: My own disposition has been to classify these cases, though of course a detailed classification is not practicable. We have, first, a group of patients featured by arrest and retarded development, the idiots and imbeciles; secondly, cases which should be grouped under hysteria, i. e., cases in which there is an undue emotional reaction and undue impressionability, and in which, due to a lessened or defective cortical inhibition, the reactions are excessive and abnormal. Again, there is another group which are dominated by the symptoms of a psychasthenia. Another group, which as Dr. Burr has pointed out, present symptoms clearly referable to mania. As regards depressive mental states, we need but recall the occasional occurrence of suicide in children. Finally, there is a group whose symptoms foreshadow a later oncoming dementia praecox.

Regarding paresis in children, my experience as a whole is in accord with that of Dr. Burr. I have encountered juvenile paresis more frequently in later years than in my earlier clinical experience.

DR. CHARLES W. BURR: As to paresis in the parents: The mother or father of three patients out of the eight had paresis. It was curious that in the generation of the children themselves, it was rare for two or more to have paresis. I have never seen two brothers or a brother and a sister or two sisters both of whom had juvenile paresis. In all these families, some of the children died in babyhood; others were still-born; others had convulsions and died; others had convulsions and were left hemiplegic and imbecilic. In not one of these eight families was any child really normal.

DR. FRANCIS X. DERCUM: One of Dr. Burr's cases, that of the child with early paranoid symptoms, suggested the paranoia originaria described by Sander many years ago. Such cases were rare, though they undoubtedly did occur.

BOSTON ORTHOPEDIC CLUB AND BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Combined Meeting, Dec. 15, 1924

C. MACFIE CAMPBELL, M.D., *President, in the Chair*

CASES TREATED BY SYMPATHETIC RAMISECTION BY DR. N. D. ROYLE. DR. BRONSON CROTHERS.

Two patients with paralysis were operated on by Dr. N. D. Royle of Australia on Oct. 29, 1924. These cases were selected from about twenty-five cases showing varying degrees of spasticity, mostly due to birth injuries, examined by Dr. Royle and Dr. Hunter. The cases selected for operation showed the typical characteristics which made Dr. Royle and Dr. Hunter feel that they were suitable for operation.

The first case was that of a boy, 8 years old, with spastic paraplegia dating from infancy and presumed to be due to a midthoracic incomplete lesion of the spinal cord. The arms were normal, and the intelligence unimpaired. A year before, he had been operated on by tenotomy of the adductors, without

much improvement. The right leg was chosen for operation as showing greater plastic tone. On the whole, this was also the less useful of the two. Since operation, the boy has had consistent muscle training and there has been distinctly more compensation than there was at the time of the operation. On the other hand, there is no evidence that the physiologic situation is in any way different from that seen before the operation. He still has active clonus, the characteristic stepping-up reflex on tapping the patellar tendon and some definite spasticity of gait. On the other hand, the leg is no longer more awkward than the other, and it may at least be argued that clinically there has been improvement which should be attributed to the operation.

The second case was that of a girl, aged 12, with a right hemiparesis presumably due either to birth injury or very early encephalitis. She was of normal intelligence and had always been able to take care of herself and get about. She had had occasional epileptiform convulsions. The whole right side showed marked atrophy, and there were stiffness and awkwardness of the right arm. The reflexes were moderately increased, and Dr. Royle was able to demonstrate increased plastic tone. Following operation this child also has had consistent muscle training, and in the opinion of those who are carrying on the treatment, she is showing rather more improvement than she did in any equal period of time before the operation. The reflexes when she was presented at the meeting were not appreciably different from those seen before the operation. The only definite statement that can be made about the case is that there is considerable increased speed in going through the various motions which make up the muscle training regime.

The conclusions seem fairly obvious that in the first place the field for the operation was an extremely limited one. Neither Dr. Royle nor Dr. Hunter feels that either of the cases reported were ideal ones for operation, and they felt that the operation might not produce a great deal of benefit. On the other hand, these cases were selected from among about twenty-five selected as being likely to meet the specifications laid down. In my opinion, the clinical results in these cases are at least inconclusive. The physiologic changes do not seem to me to be demonstrable.

DISCUSSION

Dr. A. Forbes: The physiologic basis for the procedure of ramisection is in the first place founded on the histologic observations of Boeke, recently supported by Dart, purporting to show that skeletal muscle fibers are innervated by sympathetic nerve fibers as well as by the well recognized motor neurons arising in the anterior horn cells. On the basis of these anatomic findings, Langelaan built up a theory that tonus in skeletal muscle depends on the sympathetic innervation and that the tonic contraction is different in kind from the ordinary twitch or brief contraction evoked by the medullated motor neurons. In support of this theory, he performed many experiments in which he measured the elasticity and plasticity of muscle when subjected to stretching under various conditions. The foundation furnished by these experiments for the theory seems to me wholly inadequate, consisting apparently in nothing more substantial than the superficial difference between the brief twitch resulting from a single artificial stimulus and the more gradual contraction observed in the case of certain tendon reflexes. This difference in time relations may readily be explained in the manner indicated by Liddell and Sherrington as resulting from a gradual increase in the number of constituent fibers of the muscle becoming involved in the response, without recourse to any assumption

of a dual function in the muscle fibers. Furthermore, Langelaan in seeking support for his theory of tonus as distinct from contractions evoked by stimulating medullated nerves, seriously misconstrued certain statements and observations of Keith Lucas. He cites Lucas as having found three different kinds of *contractile* substance in skeletal muscle, whereas Lucas clearly stated that he found three *excitable* substances, one of which he identified as the nerve fibers within the body of the muscle — obviously not to be classed as a contractile substance. The contraction of the muscle was the same in Lucas' experiment, no matter which excitable substance was acted on by the stimulus. Elsewhere Langelaan refers to Lucas' experiment in which he showed the stepwise increase of muscular contraction with increasing strength of stimulation as evidence of the all-or-none law of muscle response as regards individual fibers. Langelaan, ignoring the essential feature of this observation and drawing an arbitrary curve through the vicinity of the points on Lucas' graph, uses the results to prove a mathematical correlation between strength of stimulus and magnitude of response of the entire muscle, which is obviously meaningless in view of the all-or-none law. De Boer, in experiments going much more nearly to the heart of the matter, gave evidence indicating a loss of tone in the muscles of a cat resulting from the destruction of their sympathetic innervation. Bayliss' observation that heat production in decerebrate rigidity is less than in equal contraction artificially induced, has been cited in support of the theory of tonic response to sympathetic innervation. It is conceivable, however, that this economy of heat production may be explained in some way through the rhythm of the motor nerve impulses involved and perhaps also by virtue of the alternation of activity and inactivity among the individual fibers. Both Dusser de Barenne and Cobb, working independently with decerebrate cats, found no consistent difference in the rigidity between those muscles whose sympathetic innervation had been destroyed and those in which it had not. Cobb also stimulated the sympathetic fibers without evoking any visible contraction in the skeletal muscles. He further called attention to the asymmetrical influences of proprioceptive impulses from the neck muscles, reported by Dusser de Barenne, as furnishing a possible explanation of the alleged effect on the sympathetic nerves.

Hunter, in the experiments in which he found so marked a difference in rigidity between the muscles whose sympathetic innervation had been interrupted and the control muscles on the opposite side, severed the sympathetic rami some days beforehand. This procedure may well have induced vascular changes which might easily disturb the functions of the intramuscular receptors. It has been well established by Sherrington that decerebrate rigidity is a reflex which depends, among other things, on the integrity of the afferent path from the intramuscular receptors to the center. Alteration of these receptors by vascular changes may conceivably have sufficed to abolish the rigidity. Such an explanation would in no way involve the theory that tonus depends on a sympathetic motor innervation. Dr. W. B. Cannon states that he has recently observed in several experiments on decerebrate cats that rigidity is often marked in a fore limb after its entire sympathetic innervation has been destroyed. This observation confirms those of Cobb in showing that decerebrate rigidity can clearly exist in the absence of the sympathetic nerve supply of the muscle involved. Furthermore, both Buytendyk and Forbes and Cattell have published electromyograms of the skeletal muscles of a cat in decerebrate rigidity, showing the same kind of pattern of action currents that is commonly found in reflex and voluntary contraction. This evidence also tends to place decerebrate rigidity in the class of contraction evoked through the medullated

motor neurons. In decerebrating a great many animals, I have been struck by the fact that the amount of rigidity differs greatly between the two sides of the animals, probably depending in part on the postural reflex effect, already mentioned, arising in the neck muscles and perhaps also on other unknown and uncontrolled factors. In view of these large accidental variations and the evidence accumulated by Dusser de Barenne, Cobb and Cannon showing the presence of decerebrate rigidity after the interruption of the sympathetic fibers and the absence of any contraction on stimulating them, I cannot help being extremely skeptical about the entire physiologic foundation for this procedure. Even if it be ultimately proved that the sympathetic motor fibers can activate some mechanism in the muscle fiber which gives rise to a slight degree of tonic activity, it is clear that this mechanism cannot account for the strong contractions observed in decerebrate rigidity.

DR. E. W. TAYLOR: If it is true that cutting of the sympathetic rami leads to a loss of plastic tone, is it also true that stimulation of the same sympathetic fibers leads to an increase of tone?

DR. FORBES: So far as I know, this has not been demonstrated.

DR. TAYLOR: This seems to be an argument against the hypothesis which Drs. Hunter and Royle have advanced. If it be true that cutting leads to a decrease of plastic tone, it would appear that stimulation should lead to an increase.

DR. P. BAILEY: I am particularly interested in the spinal case of which Dr. Crothers spoke. The patient before the operation presented a remarkable picture of plastic tone. After the operation, in my opinion, this tone was unchanged. The statement has been repeated often that this boy was not a suitable subject for the operation. In my opinion, he was a perfect subject. According to the theory on which the Royle-Hunter procedure is based, plastic tone is a reflex phenomenon with its afferent pathway in the posterior roots of the spinal nerves, its final efferent pathway in the gray rami communicantes, and conditioned by pathways within the central nervous system extending up as high as the midbrain. Interruption of this reflex arc in any part of its course is bound to disturb plastic tone, and cutting the gray rami communicantes must result in loss of plastic tone in the parts innervated by the rami cut, according to the theory. Royle has said repeatedly, and others have repeated after him, that spinal lesions are not favorable for ramisectomy. Why did he say this? He did not say that it was more difficult to get a reduction of plastic tone by ramisectomy when there was a lesion of the spinal cord, for to have said so would have invalidated his whole theory, since ramisection in every case cuts the final motor pathway of the reflex. In order to understand, we must go back a little further. The defect in motor function resulting from lesions of the central nervous system is due to many causes: (1) loss of voluntary motor impulses, (2) disturbance of joint and muscle sense, (3) fibrosis around joints and tendons, (4) increase of plastic tone, (5) mental defect, etc. It will be noted that increase in plastic tone is but one among many disabling factors. The purpose of the Royle-Hunter procedure is to remove this one disabling factor of increase in plastic tone so that the patient may have a better chance to overcome the other disabling factors. Obviously, if his motor cortex is destroyed, if he is an idiot, or if he has ankylosis of the joints, it will do him little good to reduce his plastic tone; this is why Royle said that spinal lesions were unfavorable, for the diameter of the spinal cord is so small that it is rare to get a relatively uncomplicated increase in plastic tone from a spinal lesion.

The operation and the theory on which it is based will stand or fall on the immediate reduction of plastic tone following the operation, for this is its only indication, and for this purpose a spinal lesion is as good as any other. I say the immediate reduction of plastic tone, since plastic tone is a reflex phenomenon. That the patient does or does not have better motor function six days, six weeks or six months later is entirely beside the point, and must be explained by extraneous factors. To return to the patient; he had an evident increase in plastic tone in his lower extremities after the operation. There is, of course, the possibility that Dr. Royle missed the rami, since he was operating in a strange clinic.

Dr. Crothers: My own point of view is this: If one begins to go against accepted physiologic explanations, one is liable to get into trouble. The physiologic explanation of Dr. Royle and Dr. Hunter is a broad challenge to physiologic ideas. I should want convincing evidence before advising the operative procedure recommended. In order to get cases for examination by Dr. Royle, I went over the records of 250 cases of cerebral palsy and selected twenty-five for discussion at his clinic. From these, the two reported were chosen. The improvement in one case is definite, but not unusual; the other is entirely unchanged. Clearly the operation should at least be limited to an extremely small group of cases.

Treatment and Results of Operative Repair of the Plexus in Obstetrical Paralysis. Dr. Alfred S. Taylor, New York.

To give a foundation for the discussion of the surgical treatment of obstetrical palsy, it is necessary to review the etiology and pathology of the condition. Practically the sole etiologic factor is traction. From the anatomic distribution of the nerve roots, this traction, which is caused entirely by the separation of the head and neck from the shoulder on the damaged side, involves first and most seriously, the upper roots; and according to the degree of traction and its persistence, the roots will be damaged more or less completely from above downward. In a few instances, traction is exerted in the opposite direction, causing so-called lower arm, or Klumpke, paralysis. In a certain percentage of cases, one or more nerve roots will be avulsed from the spinal cord. In experimental lesions, this type of avulsion occurred six times in twenty experiments. In operative cases, the posterior root ganglia have been found extraspinally in a number of cases, and there has been clinical evidence of avulsion in a fair percentage of others.

The pathology will be classified as immediate, intermediate and remote or secondary. Immediate pathology consists in the tearing of the deep cervical fascia which lies just in front of the plexus and its roots; tearing of the muscles immediately surrounding the plexus and roots, especially the scalenus; tearing of the nerve sheaths, and finally of the axis cylinders themselves. In addition, there is damage to the small blood vessels, which causes infiltration with blood of the entire damaged zone. The extent of the paralysis will depend on the degree and distribution of this type of pathologic condition.

The intermediate pathologic condition results from the cicatrization of the damaged blood-infiltrated structures. This cicatrix gradually hardens and contracts and interferes with regeneration of nerves both by cicatricial obstruction within the nerves and cicatricial compression of the nerves from the outside, causing more or less permanent and complete nerve block.

Remote or secondary pathology consists in the shortening of the muscles and joint ligaments and often in deformation of the joint ends of the bones of

the extremity which result from the long continued abnormal attitudes of the extremity. Often this secondary pathology will cause a permanent handicap to the usefulness of the extremity, even after a satisfactory spontaneous regeneration of the nerve roots has occurred.

It is important to remember that the primary and secondary pathologic lesions may vary from the slightest overstretching of one of the nerve roots to complete rupture of all of the nerve roots. These lesions may occur anywhere from the origin of the roots at the spinal cord down to the formation of the distal trunks derived from the plexus. Also between the two extremes of the pathologic lesions there may be any number of gradations in the severity of the injury. The symptomatology is sufficiently typical so that little need be said. It is perhaps worth while to note that in many cases of fair severity there is primarily total paralysis of the extremity. Often when the lower roots have not been irreparably damaged, when primary complete paralysis has occurred, it is impossible to tell clinically just what the grade and extent of pathologic damage is.

In cases in which the so-called Horner syndrome is present, namely, diminished palpebral fissure, slight retraction of the eyeball, and the somewhat diminished pupil, we know that serious damage has occurred to the eighth cervical and first thoracic, either close to the intravertebral foramen or else in the form of avulsion of these two roots from the cord. After a few days or weeks, in many cases, the fingers and often the wrist will begin to move. Improvement may occur slowly but steadily over a period of two years, but this improvement may be confined to the nerves of the lower three roots. Meantime, the muscles supplied by the upper two roots may show no improvement whatever. This is ordinarily a great stumbing block to proper consideration of surgical treatment. Many men advise waiting while there is any continued improvement. Such has been the case in a number of instances in which operation has disclosed complete rupture and separation of the ends of the upper two roots.

Treatment.—My personal point of view is that treatment should follow these general lines: Primarily to place the extremity in the position which will give relaxation of the paralyzed muscles and prevent the deformities caused by the factors of remote pathology. This position, which is maintained by a brace, consists in abduction of the arm to 90 degrees, flexion of the elbow to 90 degrees, almost complete supination of the forearm and hand with extension to a straight line of the wrist, hand and fingers.

For the first three or four weeks (that is, until the evidences of traumatic neuritis have disappeared) the extremity is kept continuously in the brace. After this time the brace is removed once or twice a day for massage and manipulation, care being exercised not to pull the shoulder away from the neck during these manipulations. In cases in which spontaneous recovery is going to occur there should be return of motion in practically every one of the muscles by the end of three months, even though motion may be slight in degree in the upper arm. In certain cases which go on to reasonably satisfactory recovery, these motions may not have appeared until four or five months have passed. In cases in which recovery has been complete, the nerves have shown marked evidences of regeneration throughout the plexus at the end of three months. If such recovery has not occurred by that time, there is almost sure to be some degree of permanent defect. There is no disadvantage in waiting for six months, or even a year, if the position above described is maintained and if physical therapy is systematically used during the period.

If the recovery is far from complete by the end of six months, it is my opinion that surgical interference is indicated, although this interference may be deferred until the child is a year old, if there is reason for it.

Many men who have been interested in these cases maintain that there is no reason for surgical treatment, as those with less severe cases of paralysis of the arms get well spontaneously, and those with more severe cases are beyond help by surgical treatment. From consideration of the pathologic lesions described, it is obvious that there must be a considerable number between the mild cases in which spontaneous recovery occurs and the hopeless cases in which nothing will do any good. In the latter group, removal of the cicatricial compression from the outside and removal of the cicatricial blocks from within the nerve is sure to give much better opportunity for nerve regeneration and recovery of nerve function than mere tentative treatment with physical therapy. My experience has thoroughly convinced me that surgery has a definite place in the treatment in these cases. Exploration can be made with practically no risk to the child. A skin incision 7 cm. in length with separation of the fat pad overlying the plexus brings one down on the surgical field. Further procedure consists in the careful removal by dissection of the cicatricial tissue surrounding the roots and plexus; separation of the cicatricial adhesions between the damaged muscles and nerves, and finally by the removal of such macroscopic cicatricial lesions as involve the nerves themselves. These procedures are followed by end-to-end suture of the nerves from which portions have been resected. The wound is then closed, and the child is put up in the brace in the position previously described. The child is kept in this brace continuously over a period of three months, after which the extremity is taken out of the brace daily for massage and manipulation until such time as voluntary motion, of fair degree, has occurred. The brace is then left off more and more during the waking hours and is put on during the sleeping periods in order to prevent occurrence of the so-called remote pathology. If operation discovers no macroscopic damage in the nerves themselves, mere removal by dissection of the cicatricial tissue has frequently caused great improvement in the functional value of the extremity. If no macroscopic damage is discovered, the operation consists merely of a skin incision and separation of the overlying fat pad. This involves no danger and no risk to the child. If actual lesions are found, any risk which is involved is more than justified by the results to be derived from the nerve repair.

(A series of lantern slides were shown to demonstrate the points made in the presentation. In one group in particular in which there was total paralysis which had persisted during eleven months of the child's life, the plexus was one solid mass of cicatricial tissue which was removed and an en-to-end suture performed. This child, at the end of thirty-one months, was using the extremity, having motion in practically all of the muscles and sufficient strength to do any necessary acts. Starting from nothing, it was obvious that the entire improvement was due to surgical interference.)

Results in my group of operative cases, numbering fifty-nine, showed that in no case was the patient rendered worse by operation, except that during the period of two or three months immediately after operation there was naturally some loss from the resection of the nerves, but in no case did the patient fail to get back to at least the position he had been in preceding operation.

In addition to surgical work on the plexus, the use of the Sever operation has been of much value in overcoming the handicap due to deformities resulting from "secondary pathology." Of the fifty-nine cases, only forty-seven could

be traced sufficiently long to get results. Of these forty-seven, thirty-eight or 81 per cent., showed definite improvement. Three patients, or 8 per cent., were cured, the one extremity being as good as its opposite. Seven patients, or 18 per cent., were almost cured, having only a slight handicap about the shoulder girdle. Twenty-one, or 55 per cent., showed marked improvement; seven, or 18 per cent., showed moderate improvement. Of the remaining cases, six, or 13 per cent., showed no improvement. In three cases, the lower roots were avulsed from the cord.

Mortality: Of the fifty-nine patients, two, or 3.4 per cent., died on the table; one from hemorrhage; one at the end of the operation when apparently everything was going perfectly well and the brace and dressings had been bandaged on, suddenly stopped breathing and could not be resuscitated; one died two weeks after the operation from gastro-enteritis.

From the material presented, I am firmly convinced that there is a definite field for operative surgery in the group of obstetrical palsy cases, and that the only problem is to determine the selection of cases in which operation is indicated and the time at which it is best to perform the operation. My opinion is that the best time for operative interference is between 6 and 12 months of age. In many of the earlier cases, operation was performed at or before the three months' period; but later experience has shown that some of these patients will make such good spontaneous recovery as to avoid the necessity of operation; and since the present mode of tentative treatment eliminates most of the handicap from waiting, I feel that it is legitimate to postpone operation even up to 1 year of age. Nevertheless, many of the patients have been of ages varying up to 14 years, and even in these older patients much improvement has been gained by proper nerve repair, and this gain is much increased by the use of the Sever procedure in addition. One patient who was 18 years old, with a bad birth palsy, was operated on, but no improvement was obtained even after several years of persistent post-operative treatment.

DISCUSSION

DR. J. J. THOMAS: The subject of obstetrical paralysis has interested me for a good many years. First, in regard to the pathology of this condition. No one who has seen the plexus exposed in one of the severe cases of this type of paralysis can possibly have any doubt that the plexus has been injured, and severely, the only doubt being as to the mode of the production of the injury; and I agree with Dr. Taylor that in most, if not all, of the cases it is by stretching. The injury of the plexus is so severe that one must always bear in mind what Dr. Taylor has said, that in excising the injured portion of the plexus one must cut back the nerve cords till normal nerve fibers are found before proceeding to suture, if one expects to obtain favorable results from a resection of the plexus. The theory that these paralyses are due to a dislocation of the head of the humerus, with hemorrhage around the nerve trunks in the region of the shoulder joint, which has been advanced by Dr. Turner Thomas of Philadelphia, finds no support from the condition of the plexus seen at operation, nor can it be entertained for a moment by any one who knows the grouping of the muscles in the cords of the plexus and has observed the same grouping of the paralysis and of the atrophy of the muscles in the infants seen soon after birth when the paralysis is most marked.

These paralyses vary in extent, and we group them into the upper arm type, the lower arm type, and the total paralyses, of which the first is by far the most common. The greatest practical difficulty we have, however, in

deciding in any type of this injury as to whether a resection of the plexus is advisable in any individual case or not is due to the fact that these lesions are seldom complete, and after the original injury produced by the stretching of the plexus there is almost invariably a partial recovery. In the cases in which the paralysis at first affects all the muscles, later we may find remaining only a paralysis of the upper arm group of muscles, and even this may be partial only. Where there is fair recovery of voluntary motion in most of the affected muscles, the greater part of the disability found later comes from the secondary contractures which appear in the unaffected muscles, the opposing muscles to the paralyzed ones. These contractures are usually most marked in the subscapularis and the other internal rotators of the humerus. They limit the motion at the shoulder and in the forearm, as supination, and are the cause of the deformities of the elbow, the acromion and the partial dislocation of the shoulder which we so often see in the neglected cases as the children grow older. When these contractures and deformities have been corrected, as by the operation devised by Dr. Sever in our work at the Boston Children's Hospital, we can by the development of the weak muscles, by exercises, obtain a relatively useful arm, even in neglected cases, without resorting to resection of the plexus.

Perhaps in Boston we have used the operation on the plexus too seldom because of the good results we have seen from the use of these other methods; and because we have restricted the plexus operation to the severe cases, we have estimated its value too low. In the very severe cases in which the nerve roots have been torn from the cord, it is, of course, impossible to obtain good results from a suture of the nerve trunks of the plexus, as the remaining nerve roots cannot give good motion to all the muscles of the arm. What then should be our guide in choosing the cases in which to operate on the plexus? If we perform this operation in all cases, operating early, it seems to me evident that we shall operate in many cases in which we should get an almost perfect recovery by the use of exercises and overcoming contractures, if they cannot be prevented from forming, by operations directed to this end. On the other hand, to wait too long means that we allow the most favorable time for nerve regeneration to pass. This question probably cannot be answered absolutely correctly in every case. From my experience I should say that if we delay exploration of the plexus for a year, we are not delaying too long to get good results after a suture of the nerve trunks, while we can be sure that after the lapse of two or three years the possibility of regeneration of the nerve trunks grows less rather rapidly. I feel that each patient should be watched closely for return of motion in the paralyzed muscles and for the increase of strength in them, and if recovery is slight or after a time comes to a standstill, we should at once proceed to an exploration of the plexus, prepared to excise scar tissue and to do nerve suture to the best of our ability in that particular case. I know of no one who has had greater experience in performing these operations than Dr. Taylor, or one who can help us more in the technic of these difficult operations in young children than he, and we should all give great weight to his advice to resort to this procedure more frequently than is generally done. Of course, where this resection is required, the operations for overcoming contractures and deformities of the joints are still to be employed, if we are to get the full benefit of the improved power in the weak muscles, and we must develop them to the greatest possible extent by the use of exercises; for without proper after-treatment, all operations will fall into disrepute in these obstetrical paralyses. So the operation of

Dr. Sever still has a much wider field in these cases than any operation on the plexus, since it is of use in older children and in neglected cases; this, and other methods to overcome deformities, and the persistent use of passive movements and exercises still constitute the indispensable methods which must be employed for all of these cases in order to produce the best results.

DR. J. E. GOLDTHWAIT: The cases shown by Dr. Taylor are very suggestive and lead one to a further study. I should like to suggest that, as Dr. Taylor says, the majority of patients come to us in conditions which indicate definite neglect on the part of those who have been responsible for the child. The majority of those who have paralysis of the arms of any degree of severity have a flexed elbow, which invariably means an inwardly rotated arm. Any one with a flexed elbow will carry the arm forward. The dislocation of the humerus is largely secondary to the plexus. I, personally, feel that we should be just as much ashamed of flexed elbow as part of the development of an obstetrical paralysis as we should be of an equinus position of a foot in an infantile paralysis. Whenever that has occurred I have been impressed, in all cases we have been handling, to see how far it is possible to stretch the flexed elbow so that the hand will hang at the side with the arm straight; one may then unrotate the shoulder and improve the function there many times without having to perform the Sever operation. We have one child at present, 10 years of age, who had a typical inwardly rotated arm and pronated hand. The flexed elbow has been entirely straightened and the position of the hand, as far as inward rotation of the shoulder is concerned, has materially improved.

DR. J. S. STONE: I agree with practically everything Dr. Taylor has said, but I disagree with him on one point. He stated that in his experimental work a complete avulsion of the roots was common. In experimental work which I did a good many years ago, I found that this was rather uncommon. In accidental injury, however, I have found avulsion more common than he has. It seems to me that Dr. Taylor's cases must have been chosen with great care. Certainly when the fifth and sixth roots are completely torn across, the only logical procedure is an attempt to unite them. His cases must have been carefully selected if, as he has stated, there are no cases in which there has been an impairment of the motion which the patients already had. If these roots had not been completely torn, there must have been at least a temporary loss of motion. We have had several cases in which there has been a complete avulsion of the plexus. In some of these, no roots could be found. At times, the whole plexus has been snapped down below the clavicle. In a great many cases, suture of the damaged roots is the only logical treatment. These cases should be picked out and suture advised. Many more patients should be operated on by this method than are operated on at present.

DR. J. W. SEVER: I am very much encouraged by Dr. Taylor's paper. He has had remarkably good results; much better than any I know of in this community. I was also pleased and encouraged to find the patients that had had the plexus operations represented practically the whole-arm type, that is, the plexus at the upper roots and lower roots as well had been injured. In our experience, we find that we have as good results or very satisfactory results in these cases without any plexus operation. We have in part educated this community to bring the cases to us early, that is, during the first week or two after birth, and at the Children's Hospital we are now seeing a great many patients during the first few weeks of life. We have found the universal use of the splint which holds the arm in an abducted, elevated and outwardly rotated position very helpful, but in a certain number of cases we find that

the child cannot use the arm freely because of the fixation the splint gives. Some children began to develop certain swellings about the shoulder or about the elbow, pain and discomfort, and we could not account for them except by the constant fixation of the splint between treatments; in certain patients, therefore, we gave up the splint, and simply let the arm hang at the side; those patients have seemed to do very much better. As a result of certain discussions from Philadelphia, we induced Dr. Crothers to undertake a series of tests for reactions of degeneration in these young babies, and we found it present in all of them.

With regard to the question as to the advisability of operating in whole-arm types, the point of importance is the inequality of the pupil in relation to the injury. In general, such pupillary inequality clears up in five or six weeks, indicating that the plexus is not so badly injured as was suspected. Where it can be shown that there is a definite avulsion of the eighth cervical root and the first thoracic, there is no possible hope of repair or regeneration either by physiotherapy or operation. If it is evident that a hand has been functionally injured beyond hope, operation of any sort is, of course, not to be considered. In regard to the muscle contraction operation which we are doing at the Children's Hospital, we have found that results are decidedly better if we do not operate when we first see the case. We give the patients preliminary treatment for five or six months, massage, exercise, functional activity and work with the hand.

I should like to ask Dr. Taylor about his after-treatment in these plexus operations to find out what his period of fixation and after-care has been. The conclusions I have reached are based on the results of the study of 1,033 cases of obstetrical paralysis.

Dr. W. J. Mixter: The procedure which has been described has been attempted for many years, has been tried in many hands, and has been used perhaps ill-advisedly at times. In Dr. Taylor's hands the procedure has been used wisely. He has selected his cases far better than we have selected them during the years in which I have been interested in this subject; his technic, furthermore, is worthy of the highest commendation. His facility in handling the brachial plexus in children is very different from what I had believed could be accomplished. The application of sutures and the significance of the shortening position are matters of importance. Evidently the care of the patients before and after operation is of the greatest importance, and training is absolutely essential for good results. Dr. Taylor's suggestion that it is desirable to wait a certain length of time before attempting operation should be generally adopted. The Sever operation is unquestionably a variable procedure. Exploration should be more generally undertaken under proper conditions. If conditions cannot be improved, no harm is done. The mortality is exceedingly low, and if nothing is attempted, the disability is often great.

Dr. R. B. Osgood: Dr. Taylor asked me to speak of one case I had the opportunity of seeing. The original operation had been performed, and some improvement had resulted. There was a lapse of a good many years, three or four, when another attempt was made in this complete lower-arm type by Dr. Taylor, which resulted in still further improvement.

Dr. A. S. Taylor: Occasionally I have seen cases in which I was willing to state that there had been little or no improvement. Occasionally cases show remarkable improvement that one would not expect. No two pathologic conditions are exactly alike. One cannot group these cases satisfactorily, because they do not fall into groups. If the child is born with nerve palsy

but moves the hand or the wrist at birth; this is definite evidence that the seventh root has not been damaged. I should not consider cases in which the hand moves at the beginning as of a serious type. To my mind, the serious types are those in which there is no motion and in which one does not begin to get evidence of recovery for two or three months. One may be almost sure those patients will not make complete recovery under any kind of treatment, but they will make the best percentage of recovery if they are given physical therapy plus nerve repair. If one explores a plexus and finds nothing to be done, one has taken twenty-five or thirty minutes, has lost no blood, has learned a great deal, and the child has lost nothing. If something is found to be done surgically to the plexus, time has been saved, and whatever risk has been taken is more than justified by the prospect of doing some real good.

A number of the members in discussing my remarks stated that I picked my cases carefully. I do not know how I gave that impression, because I have taken them just as they came, and when I have advised operation it has always been on the basis of every case that came along in which I thought operation was indicated. I have operated on a good many patients when I thought operation would not result in much improvement; in the case of which Dr. Osgood spoke, the child had been treated for two and a half years for an extremity which at the end of that time had no functional value. We found that the first and eighth roots had been avulsed from the cord. Operation was the only chance of giving the arm any functional value. The child referred to is getting a certain amount of use out of that extremity. The longer the delay before operation, the more difficult the dissection becomes. If a young child is operated on when the indications are perfectly clear, say between the age of 6 weeks and 3 months, dissection is relatively easy because the cicatricial tissue has not solidified, and the operation is, therefore, very much simpler than in later cases. I am much interested in Dr. Goldthwait's discussion of flexed elbow. I cannot believe flexed elbow is the cause of dislocation of the shoulder, because I have seen a good many cases in which dislocation of the shoulder preceded flexed elbow. I should be glad to learn how those elbows can be straightened, the technic and length of time needed, since it would add materially to the value of an extremity if complete extension could be recovered.

Dr. Stone thinks I choose my cases on the basis of operating on the worst type. This is not correct. There has been no selection on such a basis; probably 50 per cent. would prove to be the upper-arm type.

With regard to after-treatment of operated patients. It has been my custom to put them in a brace which holds the extremity in the same position as Dr. Sever's, and to keep them there for three months without taking them down for a moment. I also always hold the head toward the damaged arm in order to keep it braced to prevent any strain on the nerve suture and to prevent any motion of the operated area. I keep the head in position for three weeks, because by the end of that period we have very good surgical union in those nerve stems. I also keep the brace on after the headpiece is taken off, because there will be very little, if any, evidence of nerve regeneration in the paralyzed muscles until at least three months have elapsed. At the end of that time the union would be solid, and nerve regeneration, if it were going to occur, would have advanced sufficiently far so that there would not be any increase in cicatricial tissue at the nerve point. It has been my custom after the three months have elapsed to leave the brace off during the waking time of the child and have the brace on only when it is sleeping. Then I prescribe physical therapy. Nerve regeneration itself was not sufficient to give any balance to

the muscles so that they would develop exactly the same deformities that were present before operation; in order to prevent that, I have had them use the parts during the waking hours. I have patients leave the brace off during the waking time so that by the voluntary motion they can develop to best advantage; I have them use the brace during sleeping time until the child develops sufficient power in the various muscles to render the use of the brace no longer necessary to prevent deformity.

With regard to treatment of the upper arm type in which Dr. Sever has shown splendid results with release of shoulder tissue, I think two types must be recognized: one with a good degree of motion and well developed muscles and normal looking arm but with some limitation of motion. In that type, it appears as though nerve regeneration had begun. There is another group of patients, such as the 10 year old boy I mentioned, in whom there is motion in slight degree and in whom there is some deformity but whose muscles are small. Often there is motion but no strength. It has been my experience in these cases that dissection of the plexus reveals it compressed by a large amount of cicatricial tissue which if removed, if this is possible, will give increase in strength and muscular development.

Referring to Dr. Sever's discussion, I have found that avulsion of the roots is fairly frequent; out of twenty cases, there were six in which either one or two roots had been avulsed from the cord. Six out of twenty is fairly high, and it has been my feeling that while there were a considerable number of avulsions, probably they were not more than 25 per cent. As far as I could make out from looking over my statistics, my avulsions were about 10 per cent. on living subjects. With regard to impairment, when I was speaking about those cases in which we caused no change but in which we had done no harm, I referred to the final outcome. There was a temperary loss, but in no case did we have a young child who did not regain what we started with, and in a majority of the cases a good deal more. With regard to waiting before operating, my only objection is that the dissection is more difficult. We have been carrying a good many children over a period of a year to see what could be done, before I advised operation, and in some cases one will get so far along at the end of six or eight months that operation is inadvisable. In some others, at the end of a year, one has nothing like a satisfactory condition of the arm, and operation may give immediate improvement, surely justifying the operation.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Dec. 19, 1925

CHARLES W. BURR, M.D., *President, in the Chair*

A CASE SUGGESTIVE OF FRÖHLICH'S SYNDROME. DR. B. P. WEISS.

An Italian boy, aged 10, was brought to the clinic for nervous diseases at Jefferson because of an epileptiform attack which occurred about one month before. He had had a similar seizure when he was 3 years of age. An uncle weighed about 300 pounds (136 kg.), and a sister 18 years of age had hypothyroidism and epilepsy.

The mother claimed that this child weighed 15 pounds (6.8 kg.) at birth. He started to talk and to walk at about 1 year of age. He grew progressively

stouter, and at the age of 10 his weight was 130 pounds (5.9 kg.). While under treatment during the past three weeks, he lost approximately 10 pounds (4.5 kg.).

On examination, he presented a typical skeletal overgrowth. His head was moon shaped; the hair of the scalp was dry and brittle, and it was absent at the axillary and pubic regions. The skin was smooth and appeared to have a mucoid infiltration. The thyroid was not palpable. The breasts were unusually large; the buttocks were full, and the pelvis and hips suggested the feminine conformation. There were huge folds of abdominal fat. The genitalia were exceedingly small; only one testicle was present, which was markedly fibroid in character. Whether there was a subsequent atrophy of an undescended testicle or a congenital absence, cannot be definitely determined.

Roentgen-ray examination of the head revealed a small and fairly well bridged over sella turcica. A roentgenogram of the chest showed an overdeveloped thymus. The pupils, eyegrounds and reflexes were normal. There was no polyuria or glycosuria. Serologic and sugar tolerance tests could not be performed because of the patient's unwillingness to cooperate.

The findings in this case were strongly suggestive of dystrophia adiposogenitalis or Fröhlich's syndrome. This endocrine deficiency is undoubtedly biologic in character. The small thyroid, the deficient action of the anterior lobe of the pituitary and the atrophic changes in the testicle with the overcompensation of a persisteht thymus are significant of a physiologic imbalance or dysfunction. The disturbed metabolism is due to the altered relationship of the internal secretions. One chain of glands is catabolic in action while the other is anabolic. Hyperthyroidism or overaction of the anterior lobe of the pituitary is productive of an overdeveloped and persistent thymic gland. Clinical studies have shown apparent interrelationship between the testes and the pituitary and suprarenals. When the thymus is persistent and enlarged, there is an increase of mononuclear cells which in some cases show a 50 per cent. increase.

TWO CASES OF RETINITIS PIGMENTOSA OCCURRING IN BROTHERS, ONE ASSOCIATED WITH A SPASTIC SYMPTOM GROUP. DR. FRANCIS X. DERCUM.

The first was 21 years old, and had a negative family history except that his mother, aged 48, was nervous and had a late appearing epilepsy. He weighed 10 pounds (4.5 kg.) at birth, and had had no childhood diseases except mumps; he also had tonsillitis at 10 years of age. When between 3 and 4 years of age, it was noted that he did not see well, and later he developed night blindness. Difficulty of vision increased, until at 11 years of age, atrophic changes in the eyegrounds were noted. At 12 years of age, it was found necessary to send him to a school for the blind. At 14, he began to be stiff in his legs and to have difficulty in walking. This difficulty gradually increased, until at the age of 16 he was obliged to leave the school, and at 18 the stiffness had involved both the legs and the arms, so that he was helpless. Since 18, the condition had been slowly progressive. Six months before presentation, he was able to walk a little although the gait was very stiff. Since then walking has become practically impossible.

Examination revealed marked spasticity of both lower and upper extremities. The facial expression was fixed. He spoke slowly and enunciated with great difficulty and indistinctly. The knee reflexes were elicited but inhibited, owing to the marked rigidity; clonus and Babinski's sign were absent. The biceps

and triceps reflexes were exaggerated. Sensation, sphincters, hearing, blood, urine and spinal fluid were normal. A roentgenogram of the head revealed a rather small sella but otherwise nothing abnormal; that of the chest showed an enlargement of the thymus gland. A report of the condition of the eyes follows in Dr. Hansell's discussion.

The second patient, 18 years old, had a negative history except for mumps in childhood and a tonsillectomy at the age of 7. At 10 years of age, failure of vision began, and this has gradually increased.

Examination revealed a slightly spastic gait with increased knee reflexes and an abortive ankle clonus. There was no Babinski sign. Biceps and triceps reflexes were greatly increased. Sensation, blood, urine, spinal fluid, roentgenograms of the head and chest were normal.

Comment.—In retinitis pigmentosa, there is frequently a history of nervous disease in the ancestry, such as epilepsy. Consanguinity of parents also is said to occur in about one third of the cases; it was absent in the present cases. The familial occurrence of retinitis pigmentosa is not infrequent. Often it is found associated with other nervous affections; these are numerous and varied. For example, Wilbrand and Saenger, Leber, Jendrassik, S. Solis-Cohen and others have noted, among other things, general retardation of the physical development, failure of full development of the extremities, polydactyly, talipes varus, harelip, microcephaly, idiocy, epilepsy, mental subnormality, deaf-mutism and speech disturbances. Particularly interesting are Dr. S. Solis-Cohen's cases of dystrophia adiposo-genitalis shown at the College of Physicians a short time ago. Various anomalies of the eyes, such as micro-ophthalmos, keratoconus, persistent hyaloid artery, dislocation of the lens, high grade myopia, strabismus and nystagmus have also been noted.

The present symptom group as presented by my first case has, I believe, not been described before. The spasticity is to be referred to a degeneration of the extrapyramidal pathways. It also seems probable that in his brother a similar change is beginning to take place, although the symptoms are not pronounced.

DISCUSSION

DR. H. F. HANSELL: These two cases present unusual syndromes. The eye findings are those of typical retinitis pigmentosa, namely, moderate horizontal nystagmus, clear media, atrophic optic nerves and peripheral bone cell pigmentation.

In conjunction with the report of these two patients, I should like to present the important features of the cases of four other patients, members of one family, whom I studied last winter with Drs. S. Colis-Cohen and Edward Weiss. In not one of these patients were the eye findings those of the usual or typical retinitis pigmentosa.

CASE 1.—An Italian, aged 20, complained of progressive loss of vision, and obesity. He first had noticed impairment of vision at 16 years of age, and ever since it had been progressing, until now he saw only hand movements. He had high myopia and astigmatism; night blindness was increasing each year; he had rapid, jerky vertical and rotary nystagmus augmented by attempts to see objects close to the eyes; vitreous opacities; atrophic optic nerves, retinal arteries and veins reduced markedly in size; pigment spots and a few "bone corpuscles" throughout the fundi. The fields were limited to a small area around the fixation point; convergence was deficient; the pupils were equal and reacted. Mons, girdle and mammary obesity was present. The

abdomen was pendulous, the genitalia not well developed. He had six toes on the right foot. The sella was slightly smaller than the average but otherwise normal.

CASE 2.—A girl, aged 15, complained of progressive loss of vision and of obesity. She began to menstruate at the age of 14 and had been normal in this respect. She commenced to lose vision about two years before presentation, and when last seen her vision was 20/50. Night blindness, partial optic nerve and retinal atrophy were present. Veins and arteries were contracted; numerous round pigment spots occurred throughout the periphery of the retina; the fields were contracted concentrically.

CASE 3.—A boy, aged 12, complained of progressive loss of vision and of obesity becoming more marked as he grew older. Vision in the right eye was 2/60; in the left eye, 3/60. Decided optic nerve and retinal atrophy were present. A few pigment spots were scattered in the periphery of each fundus. Night blindness was present, and he was mentally too dull to measure fields. Myopia and nystagmus were absent.

CASE 4.—A girl, aged 17, showed partial night blindness. The fields were limited concentrically to a small area around the fixation point; the pupils were dilated, the left being a trifle larger; there was no reaction to light or convergence, convergence being almost lost; there was no muscular paralysis; there were a few small vitreous opacities, one adherent to the posterior lens surface on the right. The optic nerves and retina were partially atrophied; the retinal vessels were diminished in caliber and length; pigment spots were scattered throughout the fundus; she had menstruated only once; the extremities were short and chunky, of hypopituitary type: she had tapering fingers, short hands and short arms; the feet were flat and broad, with six toes on the right foot. A roentgenogram of the skull showed nothing abnormal. The genital development was infantile.

The parents of these children were Italians; not blood related, healthy and without deformity. Of their eight children, four were thin and four fat. The thin ones were bright and had good vision, and the fat ones were dull and had poor vision.

REPORT OF A CASE OF EXOPHTHALMIC GOITER WITH A LARGE THYMUS. DR. EDWARD J. KLOPP.

This case is reported because the thymus was considerably larger than the average found at necropsy in persons who had died of exophthalmic goiter.

A white woman, married, aged 33, complained of nervousness, indigestion, vomiting, palpitation, and a swelling in the anterior part of the neck. Her menstrual history was normal. She had been married nine years, but had never been pregnant. The medical history was negative until the present illness, which began in April, 1924, with vomiting. The following day she had palpitation and rapid breathing. She then became very nervous, and remained so, and spent most of her time in bed.

Examination revealed slight loss in weight. The eyes presented all the signs of exophthalmic goiter. The thyroid was symmetrically enlarged, and a systolic thrill and bruit were heard over this tumor. The pulsation of the carotids was increased. The lungs were clear; the heart was rapid and overactive, the rate being 135 to 152. Marked tremor of the hands was present, and she was unable to hold objects.

The blood and urine were normal on admission. Later, the urine showed hyaline and granular casts. The basal metabolism was plus 45. A roentgen-ray examination of the chest was not made.

Lugol's solution was administered, and there was gradual improvement for three weeks, the pulse having ranged from 120 to 130. The nervous symptoms then increased again, and the pulse became more rapid.

On Oct. 31, 1924, both upper poles were ligated with silk, under local anesthesia. On the fourth day, there was marked improvement. Two days later, there was confusion, and finally delirium. She died eleven days after ligation. The pulse rate was 120 the day before she died.

Comment.—An enlarged thymus is found at necropsy in nearly all patients under 40 years of age who died from exophthalmic goiter. Capelle suggested that the severity of the disease was indicated somewhat by the size of the thymus. It is rather difficult to determine the exact size of the thymus by percussion and roentgen-ray examination. However, we believe that in every case of exophthalmic goiter a roentgen-ray examination of the chest should be made to determine the presence of a substernal thyroid and perhaps the size of the thymus.

Differential blood counts, as observed by Plummer and others of the Mayo clinic, do not differ materially in the fatal and nonfatal cases, if comparatively of the same age. Kocher suggested that leukopenia with relative lymphocytosis indicated a serious outlook. Shridde considered a lymphocytosis of more than 40 per cent. a contraindication to operation.

We have abandoned the old belief that the thymus begins to diminish at the age of 2. There is sufficient evidence that it increases until puberty, after which it gradually undergoes involution. Its enlargement in exophthalmic goiter probably is secondary to the toxemia, and not a cause of it, as claimed by some observers, especially those of the French school.

Occasionally there is a recurrence of the syndrome of exophthalmic goiter following resection of the thyroid. Von Haberer claims that this may be due to an enlarged thymus. Garre, von Haberer and others have recommended its removal in cases in which thyroidectomy did not afford good results. The suggestion has not been carried out by many surgeons, therefore we cannot draw conclusions on the subject. Is it possible that the thymus persists after thyroidectomy; if so, does it cause symptoms? Are we justified in doubtful cases to recommend roentgen-ray treatment over the thymus region?

DISCUSSION

Dr. B. L. Crawford: The thymus was tremendously enlarged in this case, weighing 77 gm. Aside from the finding of this enlarged thymus, the case did not present any unusual findings in the other organs. However, in addition to the thymus this woman had a very small aorta. In four other cases of enlargement of the thymus, I have observed that there was also an enlarged thyroid. A man, about 60, with symptoms of acute exophthalmic goiter was brought in for operation; he did well after operation, but later died; at postmortem, I found an enlarged thyroid and thymus. Two patients in the maternity service died following delivery, due to the toxemia of pregnancy. Both had enlargement of the thyroid and of the thymus, and both had a hemorrhage in the liver. In another case, the patient was somewhat deformed, with short lower extremities, and in this case there was enlargement of the thymus and thyroid. This patient died after a slight pelvic operation. In the structure of the thymus in the case presented, there was little stroma or fat, and comparately few corpuscles.

Dr. Francis X. Dercum: The relation between the thymus gland and the thyroid should, in my judgment, be strongly emphasized. That the relation-

ship between these two glands is close cannot be questioned; their mutual interdependence has only recently been demonstrated by Dr. Frederick Hammett of the Wistar Institute in a most brilliant manner in his experiments on rats. An overdeveloped or persistent thymus leads to an overdeveloped thyroid. It would appear that in an organism in which the intensiveness of the biologic processes of the organism is below normal, there is a persistence, first, of an excessive amount of lymphoid tissue constituting so-called lymphatic hyperplasia; secondarily, there is an enlargement and persistence of the thymus, and this in turn leads to an enlargement of the thyroid. Hyperthyroidism and its more pronounced form, exophthalmic goiter, is therefore itself the expression of a biologic subnormality. In the present case, the findings reported by Dr. Crawford point to a special persistence of lymphoid tissue in the thymus gland, which is just what we would be led to expect. Clearly, in exophthalmic goiter the enlargement of the thymus comes first, that of the thyroid is secondary. In a similar manner and under somewhat different circumstances, an enlargement of the thymus leads to an enlargement of the anterior lobe of the pituitary, and thus results in acromegaly.

PATHOLOGIC SPECIMENS, WITH HISTORIES OF CASES. DR. S. F. GILPIN.

CASE 1.—*Tumor of the Pons.*—A white man, aged 30, complained of deafness in the right ear, lachrymation in the right eye and weakness of the right side of the face. The family and medical histories were negative. The present trouble began with vertigo three months ago. Shortly after this, he noticed weakness of the right eye, deafness of the right ear and loss of power in the right side of the face. He had double vision and headache at intervals. The vertigo was so severe as to interfere with walking, and was aggravated by any movement of the head.

Examination revealed that the pupils were equal and reacted to light and in accommodation. He was unable to close the right upper lid, and the right side of face was partially paralyzed. The tendon reflexes were markedly increased, but no Babinski sign or clonus was present. He had no tremor of the hands or tongue. Temperature and respiration were normal. The pulse rate varied from 80 to 120.

Vision in the right eye was 20/30; in the left eye, 20/20. Tension and muscle balance were normal. In both eyes, the mediae were clear, the retinae hyperemic, the nerve heads redder than normal. No gross lesion was seen. There was fine, slow, lateral and rotary nystagmus. A roentgenogram of the skull showed a widened sella turcica with flattening of the anterior clinoid processes. The Bárány test revealed an end-organ lesion on the right side. Nose and throat examination, blood count, urine and Wassermann tests were negative.

After six weeks, the patient left the hospital, with slight improvement in the seventh nerve palsy but no other change. He returned in five months much worse, complaining of headache and showing in addition to former findings, a right sixth nerve palsy, marked ataxia of station, great difficulty in walking, with staggering from side to side. The voice was hoarse. There was no loss of power in the extremities. Some stuttering was noted when he talked for any length of time. The tongue was protruded straight. The pupils were equal and reacted normally. The knee reflexes were greatly exaggerated and equal. Ankle clonus was present but no Babinski sign; no wasting of muscles had occurred. The eyegrounds were normal. He developed cardiac and pulmonary difficulties, and died five days after admission.

Pathologic Report.—The meninges were normal. On removing the brain, it was found that the base of the brain, especially the pons, was distinctly flattened. The medulla was normal. The pons measured 5 cm. across the base. The basilar vessels were deflected to the left, and the right side of the pons was much larger than the left. The right side of the pons encroached on the anterior surface of the right lobe of the cerebellum. On section through the pons, the tissue was a homogeneous gray. No difference in the two sides was recognizable. On section, the remainder of the brain showed no evidence of gross lesion. The ventricles were not dilated.

Histologic Examination.—Section from the pons showed practically the entire right half to be composed of cellular tumor tissue. The cells varied in size and shape, with comparatively little intercellular substance, which contained many small blood vessels. . The line of demarcation between the tumor and normal tissue was not sharply defined.

Diagnosis.—Glioma of the pons was diagnosed.

Comment.—The sudden death of the patient prevented an operation for a cerebellopontile angle lesion. As we look at the specimen, we are satisfied that we did not add to the mortality list of the surgeon. It is interesting that with such an extensive lesion the patient's optic nerves should remain normal.

Case 2.—*Cerebral Hemorrhage with Convulsions.*—A white man, aged 52, whose family and past histories were negative, denied venereal infection and admitted moderate use of alcohol. While standing in his bathroom, he suddenly developed a convulsion with unconsciousness. He was prevented from falling, and did not strike his head. The convulsion was generalized, with marked tonic followed by mild clonic movements. He ground his teeth and frothed slightly at the mouth. There was no facial asymmetry. The right pupil was dilated and did not react; the left was moderately contracted and reacted sluggishly to light. The reflexes were increased and equal, without clonus or Babinski sign. The blood pressure was: systolic, 140; diastolic, 90. The pulse rate was 84 and strong. The attack lasted twenty minutes, and the return to consciousness was slow and associated with mental dulness. The following day he had a similar attack, and the next day he had four more. One attack was seen by Dr. Burns, who noticed the seizure beginning in the left arm.

We believed the patient had a thrombosis in the right side of the brain producing cortical irritation. Eye examination revealed a moderate grade of sclerosis of the retinal vessels. The convulsions stopped after five days. The patient was dull, confused and violent at times; he gradually became worse, sank into stupor, and died on the tenth day.

A roentgenogram of the skull showed nothing abnormal. The urine showed a trace of albumin. The spinal fluid was bloody and the Wassermann test of the fluid was negative.

Pathologic Report.—On removal of the calvarium, a large blood clot was noted adherent to the dura over the left parietal region. The dura over the base appeared normal. The sinuses were normal. The convolutions in the frontal lobes were flattened and the sulci shallow. This condition was present though not so marked in the convolutions and sulci of the left occipital lobe and the right hemisphere. The vessels were distended and hard. There was a large amount of jelly-like clot over the cerebellum. On incision of the corpus callosum, considerable bloody fluid was present in the ventricle. On section, a large area of hemorrhage and necrosis was noted in the left hemisphere; it extended from the cortex to the lateral ventricle in a wedge-shaped

area; the base was formed by the superior and middle frontal convolutions, the apex ending in the left lateral ventricle. The hemorrhage extended from a position 4 cm. behind the tip of the frontal lobe to the fissure of Rolando, involving the optic thalamus, the caudate nucleus, the upper limb of the internal capsule and a portion of the gray matter. The island of Reil was not involved.

CASE 3.—*Multiple Brain Tumors.*—A white boy, aged 10 years, was brought to the hospital, with complaints of headache, vomiting, convulsions and stupor. The family history was negative. The patient had had a mastoid operation at the age of 8 months. At the age of 6 years, he had had spells at intervals of six months during which he ran screaming about the room. Later, he lay apparently unconscious, and his left hand would twitch for one to three minutes. He was given phenobarbital for these attacks, and they disappeared; he was in good health until his present illness.

His present illness began two months before presentation, with severe frontal headache. About the same time, he began to vomit after eating. In two days, he became comatose and was taken to the University Hospital, where he regained consciousness in three days, and in three weeks he left the hospital. Two days after his return home, he was again taken with vomiting and severe headache, and returned to the hospital. He again improved and went home, Sept. 7, 1924. At home, he felt well for a day or two, when he was again taken with severe headache and vomiting. This kept up for one-half hour, when he had a convulsion. This lasted for a short time, when he again complained of headache. Since then he had been stuporous and had had incontinence of bowels and bladder.

Examination revealed a well nourished boy, dull and stuporous, who slept most of the time. When awake, he was irritable, and at times cried out with pain in his head. His face showed a complete palsy of the left side. The pupils were equal and reacted promptly to light. There was a suggestion of stiffness of the muscles of the neck. The abdominal reflexes were present. Gait and station were ataxic, and he usually tended to fall backward. There were slight knee reflexes, without a Babinski sign or clonus. Kernig's sign was positive.

Eye examination revealed clear media, hazy disks, blurred edges, full and tortuous veins. There were no hemorrhages or white spots. Three weeks later, suggestive atrophic changes secondary to neuritis were noted. The arteries were contracted, the veins tortuous but not unduly dilated. The Wassermann tests of the blood and fluid were negative. The spinal fluid showed from 1 to 3 cells. The white blood count on September 14 was 6,700; on September 21, 9,100; on October 12, 23,000. Urine was not obtained because of incontinence.

Roentgenograms showed large calcareous masses in the head, apparently on the right side — one in the temporal region and the other in the occipital. The largest one was in fairly close relation to the occipital and parietal sutures, and the smallest one was also fairly superficial and in the temporal fossa. In addition, it was evident that there was markedly increased intracranial pressure, as the sutures were considerably separated, and the convolution markings exaggerated.

Course and Outcome.—September 14: There had been no more vomiting since the patient had been taking liquid diet. He was quiet most of the time, although at times he cried out with headache. He was comatose; the temperature reached 106 F. by axilla; the deep reflexes were increased, and Babinski's

sign was present. Lumbar puncture yielded a bloody fluid. The patient died twenty-six days after admission to the hospital.

Pathologic Report.—On removing the calvarium, the lines of suture of the bones of the skull were prominent. In the right temporal region, extending from just to the left of the midline along the fronto-occipital junction down to the right temporal bone, the skull was very thin, showing numerous depressions on the inner surface, but the inner plate was not destroyed at any point. On removing the dura, the pia-arachnoid was found to be edematous. Over the entire brain, the superficial vessels were injected, but no evidence of inflammation was observed. The blood vessels at the base appeared normal. Scattered over the surface of the brain were numerous circumscribed, firm, nodular areas, in places extending slightly above the surface of the brain and following the convolutions. The areas were firm and slightly lighter in color than the surrounding brain tissue and in some places elevated. The largest of the nodules was on the under surface of the right temporal lobe, and while they were scattered over the entire surface of the brain, except the cerebellum, they were more numerous in the right frontal and parietal lobes. Both the lateral ventricles were markedly dilated, and a soft, red, hemorrhagic cystic nodule was found in the left choroid plexus.

Pathologic Diagnosis.—Multiple circumscribed lesions were found in the cortex of the brain with marked thinning of the skull in areas and hydrocephalus. Sections from the nodular areas in the brain cortex stained with phosphotungstic acid hematoxylin showed a rather diffuse gliosis near the surface, the tissue containing few cells. At the margin, the lesion gradually faded into the normal tissue. No inflammatory reaction was observed. Sections from the nodule in the choroid plexus showed numerous dilated blood vessels with a considerable proliferation of the endothelial cells, some of which were multinuclear. The diagnosis was glioma.

Many symptoms of this patient suggested epidemic encephalitis with meningeal symptoms. There was no adequate explanation of his temperature, which was irregular and not characteristic of any special infection. In one twenty-four hours the temperature ranged from 97 to 106 degrees. The only postmortem explanation appears to be that of increased intracranial pressure.

Case 4.—*Multiple Brain Tumors: Tuberculoma.*—A man, aged 34, was admitted to the hospital, Aug. 29, 1923, and died Sept. 10, 1923. He complained of headache, vertigo, vomiting, interference with sight, smell and hearing. His present trouble began ten months before presentation, with severe, constant, frontal headache, not relieved by drugs. Headache and vomiting continued. Two months before admission to the hospital, he developed deafness, dimness of vision, peculiar and terrible smells and marked vertigo which almost prevented walking. He had difficulty in speaking, masticating and swallowing.

Examination revealed dilated pupils; the left did not react to light or in accommodation. A partial paralysis of his left sixth and seventh nerves was noted. Deafness was present in the left ear. There were choked disks in both eyes; an active, acute process was found. Trouble in speaking was marked, and he could not protrude the tongue. There was no evidence of lesions of the heart or lungs. The liver was enlarged. The knee reflexes were present; there was no clonus or Babinski sign. On standing, extreme vertigo developed, and he was unable to walk.

A roentgenogram showed moderate increase in intracranial pressure, as shown by spreading occipital suture and a few convolution markings. The blood and urine were normal.

Ten days after admission, the patient arose during the night, suddenly collapsed and died.

Pathologic Examination.—Several firm nodules were discovered in the lungs just beneath the surface; the centers of some were necrotic. The left suprarenal was firm and nodular, and one half of the substance was replaced by firm, grayish tissue, which was sharply defined at the edges. The right suprarenal was normal. The tumor in the liver was practically one growth, nodular in formation, 17 cm. in diameter (longest); the tissue was broken down at the center and soft and yellowish.

The dura of the brain was normal. There were two firm, nodular masses on the under side of the brain; one was in the anterior portion of the lower surface of the left temporal lobe, and the other involved the posterior surface of the left lobe of the cerebellum. A similar smaller nodule was found on the under surface of the left occipital lobe. The nodules were firm in consistency and sharply circumscribed. After fixation in liquor formaldehydi and on section, the nodules were solid throughout and gray, and shelled out of brain substance readily. The ventricles were dilated. Sections from various masses showed extensive central necrosis, marginal fibrosis and mononuclear leukocytic infiltration.

Diagnosis: Tuberculoma was diagnosed.

The patient presented a history of "peculiar and terrible smells" to use his expression. One tumor in its growth had irritated and pressed on the under surface of the anterior part of the left temperosphenoidal lobe.

A Case for Diagnosis Featuring Ascending Pyramidal Unilateral Tract Degeneration with Other Associated Symptoms. Dr. M. A. Burns.

A woman, aged 40, married, had as her chief complaint stiffness and heaviness of the left leg. Her family history was negative. The patient had pleurisy in 1922, after a delivery followed by a questionable tuberculosis, from which she recovered. Menstruation began at the age of 13 and had always been regular and moderate. There was no history of abortions or miscarriages, and she had had eight normal pregnancies. Thirteen months before presentation, she began to notice numbness and weakness in her left leg, and later she complained of a similar sensation in her left arm. Then her left leg became stiff; this came on gradually and varied in intensity. After her leg became stiff, she complained of her arm, and at times she could hardly hold a comb. Any psychic disturbance made her worse.

Examination revealed a well nourished white, adult female. Her scalp, ears, nose and throat were grossly negative. Station showed a positive Romberg sign. She held the left leg stiff, but there was no evidence of definite spasticity or flaccidity. The pupils were equal, somewhat irregular, and reacted rather sluggishly to light. The tongue was clean, protruded in the midline and was not tremulous. The teeth were in fair condition. The tonsils were large and cryptic. The thorax was normal in outline. Signs suggestive of pulmonary tuberculosis were present at the left apex. A weakness of the lower half of the left side of the face was noted. The grip was better on the right than on the left side. The left biceps reflex was slightly increased. No definite spasticity was present in the lower extremities, but power was slightly decreased on the left side. There were no sensory disturbances. The right patellar reflex was normal with a Babinski sign but negative for ankle clonus. The left patellar reflex was increased. Ankle clonus and Babinski sign were present.

Three weeks after admission, a slight ptosis was noticed on the left side, with a more definite weakness in the lower part of the left side of the face.

Findings in the right eye were: media clear, disk good color, central cup, margins fairly clear (moderate astigmatism). The vessels were normal. The report on the left eye was the same.

A roentgenogram of the head showed the coronal suture not well united. There was marked hypertrophy of the posterior clinoid processes on both sides, but no evidence of increased intracranial pressure. Roentgenograms of the lumbar and dorsal spine revealed no evidence of disease.

The blood, spinal fluid, blood Wassermann test and urinalysis were negative.

DISCUSSION

DR. CHARLES K. MILLS: I first presented a case of this type to this Society some time ago; the case I described was somewhat similar to the one now presented. I subsequently wrote several papers on the subject. One condition which resembles it closely is a unilateral ascending paralysis agitans. A slowly developing brain tumor in the motor region may also resemble this affection, but usually it can be differentiated by the presence of the general symptoms of brain tumor.

TRIPLEGIA APPEARING EIGHTEEN YEARS AFTER AN INJURY OF THE HEAD AND GREATLY BENEFITED BY OPERATION. DR. C. FRED BECKER.

A man, aged 58, had a negative history except that he had had an injury of the head eighteen years before; at that time he received a severe blow on the left parietal region rendering him unconscious owing to a cerebral hemorrhage. Operation was performed, and he recovered completely. His complaints when presented were (1) total disuse of both legs, more marked on the right, (2) some stiffness and weakness of the right arm.

In July, 1924, he began to feel stiff and weak in his right leg, and a few weeks later noticed that the right arm felt weak and stiff. In about eight or ten weeks, he was unable to walk without support.

Examination revealed that he could not stand alone. His gait with support was spastic, more marked on the right. The pupils reacted well to light and in accommodation and were equal and regular. There was no nystagmus. The right arm was somewhat spastic. The right biceps and triceps reflexes were increased; the left apparently was normal. Grip was poorer in the right hand. Both legs were spastic, the right markedly so, with ankle clonus and Babinski on the right but none on the left; the knee reflexes were exaggerated. No atrophy, sensory disturbances, muscle fibrillations or tremors were noted. A large, irregular area in the left parietal region pulsated but did not protrude. There was no speech defect. The patient was oriented, cooperative, answered questions readily and apparently rationally. Memory was good. No delusions or hallucinations were elicited.

The blood, spinal fluid and eyegrounds were negative. A roentgenogram of the head showed evidence of an old operation and thinning of the posterior margin for about an inch.

Operation was performed at the site of the old scar, revealing excessive scarring of the scalp and dura and a markedly thickened dura with firm adhesions about the old wound and at the margins to the brain tissue. The dura was opened in center of the field, about two tablespoonfuls of fluid escaping. The brain tissue beneath was edematous in appearance.

Four days after the operation, free movement of the right arm and partial movement of both legs was possible; seventeen days after the operation, he was able to elevate, flex and extend both legs freely in bed, and spasticity was decreased. Babinski's sign was absent, and slight ankle clonus was present. To date 50 per cent. recovery of the use of the legs has taken place.

DEMONSTRATION OF SOME IMPROVEMENTS ON THE SUPPORT USED IN FACIAL PARALYSIS. DR. N. S. YAWGER.

It is now about four years since I demonstrated before the Society a method supporting the tissues in facial paralysis through the means of adhesive plaster, and during the last two years I have been able to simplify and otherwise improve the device.

Whereas the support formerly consisted of three pieces, I have reduced the number to two, because I now employ a substance distributed under the trade-name of "Tirro"; the substance is very durable and adheres most tenaciously. "Tirro" by reason of its rubber content is water-proof, so that men in shaving around the device find that it does not loosen. But after having served its purpose, the matter of its adhering so intimately renders removal difficult. Many different solvents were tried, and finally it was found that carbon tetrachlorid would answer the purpose admirably. A few drops of this liquid applied with a medicine dropper at the upper edge of the support causes it to loosen at once. After an extensive use of these substances — "Tirro" and carbon tetrachlorid — I do not fear dermatitis as a sequel.

When properly applied, the device offers a number of advantages: There is less lachrymation and conjunctivitis. The patient can immediately eat and talk more satisfactorily. By holding the tissues where they belong and by preventing the intermittent tug from overaction on the opposing side, the course of the disease is shortened, and some of the contractures are prevented.

NEW YORK NEUROLOGICAL SOCIETY

Annual Meeting, Jan. 6, 1925

EDWIN G. ZABRISKIE, M.D., *President, in the Chair*

ABNORMAL TONGUE MOVEMENTS FOLLOWING ENCEPHALITIS: REPORT OF A CASE. DR. IRVING H. PARDEE.

A boy, aged 17, had a fall in January, 1920. A sled knocked his feet out from under him, and he landed on his head. Following this he was in bed for some time, and was said to have been "out of his head." His eyes were examined following the accident, because of some visual disturbance, type not described. For six months there was considerable disturbance of sleep, tending to reverse the sleep mechanism. The patient tried to go to school, but could not sit still. For two years afterward, when sitting down, he would lean forward with head projecting. More than a year ago, he had chewing-like movements, and the present abnormal tongue movements began last spring. During the summer, the voice became altered, so that he now complains that when he talks "only wind comes out, but no sound." The mouth would open and the tongue protrude; this was soon followed by rhythmical, irregular, choreic-like

movements, which have persisted. The father stated that the boy's entire personality had changed; from being a bright and alert boy, he is now slow to respond, and his movements are very much retarded.

Physical examination revealed the head projecting forward, with a slight, generalized attitude of flexion. The gait did not show festination; but a marked loss of associated movements was noticed in the left arm. The movements were all somewhat slow. The facies showed a lack of emotional play approaching the mask of Parkinson. There was slight fixation of the right side of the face. The left pupil was irregular and sluggish to light; the right was regular and reacted. There was a tremor of the left arm of a rhythmical oscillatory type, more marked on rest.

The movements of the tongue I have called rotary and extension of an almost rhythmical choreo-athetoid type. Their exact quality almost defies description. They were almost constant, ceasing only when interest was centered on something very intently.

Neither ataxia nor dysmetria was present; there was no alteration in the reflexes. The fundi were normal. A roentgenogram of the skull showed no fracture.

DISCUSSION

Dr. E. L. Hunt: It seems to me that the evidence points strongly to encephalitis. The condition as one of the sequelae of encephalitis is an exceedingly unusual one. I have found reports of the same condition occurring in foreign cases—four from France and two from Germany. I have never known of a report of a similar case in this country. One of the cases reported abroad was just the reverse of this one: the tongue remained out longer than it remained in, but it was constantly in motion.

Dr. I. Abrahamson: There is no question about this being a sequel of encephalitis. We have seen these tongue movements, but not so marked as in this case. I have seen also lateral movements of the tongue and movements of the jaws, with or without movements of the tongue. A boy now in the Montefiore Hospital presented an opening movement of the jaw, occurring in episodes. That boy's left arm had the same position as that in the case shown. The tongue movements I have seen in at least six cases; but in no case was it as marked as this. A point of interest is the absence of salivation, in spite of the constant inward and outward motion of the tongue. One would expect that salivation would be stimulated by this. Is the boy getting any scopolamin or belladonna, or something that stops salivation? I have an idea that he will lose the movements of the tongue, as most of the other patients did. The boy at the Montefiore Hospital still has some of the movements of the jaw, but they are becoming less frequent.

Of interest also is one other point. Patients with ordinary parkinsonian movements become steadier when they "intend," but in those with encephalitic parkinsonian disease, the movements become worse. If the boy had ordinary parkinsonian movements, he would halt, and then start and get through his sentences. This boy has the greatest difficulty in talking, and this increases the movements of the tongue just as the patient with postencephalitic parkinsonian disease increases the movement by intention. This movement resembles a choreo-athetoid movement of the tongue. Occasionally, in chorea, when the patient thrusts out his tongue, he will get his jaws on it and will bite his tongue; but in this patient, we have just an isolated tongue movement, without any movement of the jaw. The tongue always stays in a straight line in the

middle of the lower jaw. The tongue movements are accompanied at a certain level of integration by movements of the lower jaw. Here there is no lower jaw movement. Did the tongue deviate to the left?

DR. PARDEE: No; it is in the midline.

DR. ABRAHAMSON: The level of integration must be at a different level from that of the tongue acting in conjunction with the lower jaw.

DR. I. S. WECHSLER: There is not the slightest doubt in my mind as to the diagnosis of encephalitis, although it is interesting to speculate on its relation to the trauma. That could come up for decision in case of litigation, but I feel it has nothing to do with the present case. I should like to point out the significance of the patient's tongue movement in view of the accepted anatomic localization of the lesion in postencephalitic Parkinson movements, and the fact that jaw and tongue and champing and chewing movements have been associated with lesions of the substantia nigra. It seems rather strange that patients with encephalitis who show the Parkinson syndrome show so few tongue and jaw movements. This boy has both the Parkinson syndrome and the tongue movement which can be correlated with the pathologic lesion in the substantia nigra, and the experimental physiologic evidence adduced by Bechterew and his assistants.

DR. CHARLES BERNSTEIN: I have seen three of these tongue cases. A boy, about 20, protrudes, rolls and chews his tongue. A boy, about 16, makes almost the same motion of the tongue as this patient. All three patients were feeble-minded; all had postmeningitic cases.

DR. PARDEE: I have nothing more to add, except that I believe the element of trauma in this case has nothing to do with the later developments. I have seen previous cases in which trauma seemed to usher in an encephalitis, just as we see trauma as a predisposing factor in various types of disease, such as general paresis, and so forth. I have under my observation now a legal case in which a parkinsonian syndrome, the result of encephalitis, began immediately after a trauma. Trauma is thus a factor about which one can only speculate.

THE NEW YORK STATE PROGRAM FOR THE CARE OF THE MENTALLY DEFECTIVE. DR. SANGER BROWN, 2nd.

Through various surveys in the past, we are now in a position to state roughly the number of mentally defective in New York state, and therefore to outline their needs. The surveys indicate that there are at least 40,000 mentally defective in the state outside of institutions. We do not need to put all defective persons in institutions, but we do need institutional accommodations for about 10,000 of them. We now have accommodations for less than 6,000, so we need much additional construction.

It may be interesting to give a review of the history of the care of the mentally defective in the state. The care of the mentally defective in New York state was first undertaken in 1851, in Albany. A new institution was built two or three years later at Syracuse, where there are now accommodations for about 700 patients. The first phase may be called that of special training. At that time, Dr. Edward Seguin arrived in this country from Paris, much interested in special training of defective persons as it had been undertaken in France. He became associated with this work at Syracuse. Dr. Seguin had the idea that if defective children were properly and carefully trained, they could be brought up to normal intelligence, or at least that their intelligence could be increased. While this has proved to be contrary to the facts, never-

theless the methods which were instituted at that time in the way of manual education and special sense training were the foundations for the training of the mentally detective in this country.

The next phase in the care of the mentally defective was a eugenic plan. The second institution was started at Newark, N.Y., its purpose being the segregation of women in the child-bearing period. This was opened in 1878.

The institution soon became full, so that comparatively few new patients could be admitted from year to year. This plan therefore was of limited value in itself. The next institution was opened at Rome, N. Y., for chronic low grade cases of custodial type. In 1907, the institution at Letchworth Village was planned, construction being started somewhat later. This institution has developed along modern lines, which are more comprehensive.

In the administration of these institutions, there are three main types to care for: (1) the school cases — children of about the type which we get in the ungraded classes in the public schools; (2) adults requiring segregation and institutional care; (3) the crippled and infirm who require nursing. In the state organizations, more recently the institutions for defective delinquents has been added — the institution for males at Napanoch and the division for defective delinquent women at Bedford.

In these institutions for the mentally defective, the training policy plays an important part. About one third of the inmates are children of school age, to whom special training is particularly applicable. It is for this reason that these institutions are called state schools for mental defectives, and a teaching staff with a head teacher is provided for this work. The other main features of the institution are the psychiatric work and the general medical supervision.

From these schools has developed the colony system as inagurated by Dr. Charles Bernstein of Rome, N. Y. This is a new development in this country in the care of the feebleminded. He places boys out to work on farms in colonies. For girls, he rents a cottage in one of the factory towns, and places the girls there under supervising matrons. Many such colonies have been started throughout the state.

In addition to the institution work, therefore, with its colonies and parole cases, is the work directly under the commission. Clinics have been started throughout the state for diagnostic cases, there being at present fifty-three in all. Five social workers are directly under the commission to supervise these clinics and to carry on the after-care work.

In summary, then, the present state policy consists of a combination of those methods which have been worked out in the past and which have proved satisfactory, and is as follows: development of institutions for the care of all types requiring it; special training for proper cases in the institutions; segregation of other types; social rehabilitation through colonies and parole as far as possible; diagnostic clinics throughout the state connected with the public schools and courts; social supervision by field workers; full use of the special classes in the public schools; special institutions for defective delinquents.

DISCUSSION

DR. CHARLES BERNSTEIN: Dr. Brown has been very active in helping us to a more useful and more practical program, and has helped us in our point of view of problems which ten years ago everybody approached from other points of view. The physicians were indifferent to the problem of feeble-mindedness, and left it to the social workers and psychologists and women who never had, and knew little of, children. Surely any one who observed

the work closely twenty years ago saw this situation. Another aspect of the problem is that they were asking the state to appropriate more and more money for palatial structures in which to place the feebleminded in custody for life. The only ideal was to duplicate the system of state hospitals for the care of the insane. This was going on under the urging of the eugenists, who felt that this was the proper method. We found that these people were efficient, doing the work in institutions, and we found that we had more people in the institutions who were able to work than could economically be kept occupied. They were just as troublesome in the institutions when unoccupied as outside, and sex perversions prevailed. There was a lot of human energy pent up which would surely crop out abnormally somewhere unless otherwise consumed or relieved. As the work developed, we saw that there were two situations wherein there was great demand for help; one, agricultural work for boys, and the other, domestic work for girls. We found that we could send the boy to a farm and the girl to do domestic work, and that many would be just as well thus supervised. Probably we could hold them longer under supervision than were they held indefinitely in custody in the institution. We can train them better to succeed in the world as a result of such parole and extra institutional supervision, and thus carry out a more practical eugenic program than by any system of life custody yet devised, principally because we can care for a much larger number and guide them in an economic as well as humanitarian way for a much longer period; in fact, as long as seems necessary in each case. When the girl's histories are looked up, we find that they are leading fairly decent lives, and most of them are socially and economically efficient. They or their relatives wanted the money they earned, but later we were fortunate enough to get the legislature to give us control of their earnings. Mr. Davis, who is working with the State Charities Association, brought out the point that we were stabilizing the feebleminded. We do know that these feebleminded people will form good habits of life, and if they repeat them for years in succession, they will nearly always retain them. I know that some of these feebleminded are more stable-minded than some of my unclassified friends. As a result of these activities, we are carrying 890 people in colonies — twenty-two farm colonies with 450 boys, and fourteen colonies with 440 girls — and 500 people on parole. We find that when Johnnie is 14 years old, he gets no farther in formal school; he is incapable of further school progress and is becoming troublesome to handle and supervise because of larger energy and interests which must be usefully liberated and directed along manual and industrial lines. The idea is to place him where his energy can have an outlet. We place from twenty to twenty-four boys on a rented 100 to 200 acre farm, with a farmer and his wife who have had experience in institutional training as well as in farm life, and who understand how to handle these boys. Girls are sent to the colonies in towns to do domestic work in small average grade families. It is our policy to approach this problem in a human way, and we can thus advance a strong eugenic program and carry it out with the feebleminded, especially as sterilization and other extreme measures universally fail. I know of three women on whom sterilization was performed, in whom the results tend to support the present public attitude toward sterilization as a failure and an impracticable measure. One typical case is that of a girl of a wealthy family, who could not be kept off the streets. She was sterilized, and is now passing syphilis along. The English commission that studied the subject of sterilization came to the conclusion that probably the second or third generation would suffer more from degeneracy as a result of ravages of syphilis and venereal disease than

they would from the feebleminded offspring of such unsterilized feebleminded. Another aspect of the problem is that the great majority of idiots and low grade imbeciles who might be reached through sterilization measures are low grade organically, and would never reproduce. They are not the hereditary type with possible hereditary transmission, and really constitute only 20 per cent. of the feebleminded. We are dealing now with 80 per cent. morons and 20 per cent. idiots and imbeciles. I believe the moron is a normal part of the human race in the process of evolution. We have had some experience in support of this at Rome. We have been taking care of feebleminded women who are pregnant or who have a small child. We have had 250 of these children in the last ten years. We have had also 400 morons, who have left our institutions and have married. Our field workers are studying these cases. There are enough data to show that the so-called family condition of mental degeneracy does not exist. It is really regeneracy. We may not have offspring of much greater intelligence than their parents, but we will have more stabilized and better trained persons, and we need stabilized persons (morons) to do the common work of the world. Our morons must grow our vegetables and food for us, and this they may do and remain at the same time in a simple rural environment, which is not too complicated for them, and in which they are happy and contented. The brighter people will not stay on farms and do the hard work. The interesting thing is that if we place the stabilized moron and keep him in a simple environment, as on a farm, he is just as much a fixture there as the dog or the mule. The farmer likes the moron, and the moron likes the farmer.

That is the problem as I see it today. Approach it from a common human standpoint, and we may solve it.

We know what Nature does with organs or functions which are no longer needed, and I am sure she will thus deal with the moron when such a person is no longer useful. We were told that the moron was a prolific reproducer. We have had experience enough to know that that is not so. The imbeciles in our experience are the prolific reproducers. Among the 400 women who left Rome who married, not one had more than four children, and they averaged less than two children per family, over one-half of them having no children. Possibly training does for the moron what it does for higher grades of society. We know that the graduates of Harvard, Yale and Vassar produce fewer children than the average family. There is apparently something organically basic here. I know the birth control people have not helped the morons who have passed under our observation.

Dr. L. Pierce Clark: The excellent work Dr. Brown has done in getting this problem into the field of medical sociology should be appreciated by all of us. It places the problem in a humanistic light.

Dr. Bernstein's work is almost epoch-making in handling the moron and the placing of the final product. It is unfortunate that medical men have in many instances confined themselves so closely to medical pathology, and have not taken cognizance of the fact that mental defectives cannot be properly handled in private families. It is unfortunate for the institutions that they do not correlate their functions sufficiently so as to take into account these human beings. The problem will remain in the hands of very special workers, and we suffer in not correlating a larger number of the mental diseases of this branch of neurology.

Dr. Joshua Rosett: Will Dr. Bernstein please explain what he meant by saying that the moron is a normal part of the human race in a process of evolution?

DR. BERNSTEIN: I mean that the processes of evolution have not gone on to perfection in a great many lines; by-products useful for the time are frequently evolved, and Nature produces pretty nearly what she needs as human beings to carry on the work of the world. There was a time when people of a higher grade of intellect were willing to stay on the farms and work and go into the factories. They do not do it any more. Somebody must do the common work of the world. We know that Nature takes care of such situations, and that is why we think that so long as Nature needs the moron (and Nature needs more morons than ever before) we shall have them. We see this principle evidenced in the birth rate. In Germany, within three years after the war, there was a larger birth rate of males than females — Nature's need to balance the sexes in the interests of progress.

DR. BERNARD GLUECK: In order to appreciate fully the significance of the state's program with respect to the mentally defective of this state as outlined so ably by Dr. Brown, one has to recall what the situation was only twenty-five or so years ago. When one remembers how misunderstood, neglected and abused these unfortunates were in the past and that even today in some parts of the country there is practically no special provision for the case of these handicaped people, we have just cause to be proud of the manner in which the problem is being dealt with under the able supervision of Dr. Brown. He has told you that we have about 40,000 defective persons in this state. I think that if one were able to take into account the various social institutions, such as prisons, jails, and reformatories in which undoubtedly large numbers of defective persons are housed, the problem would probably be found to be much more extensive than it appears from merely studying the situation in what are primarily medical institutions.

There is no doubt that the progress in dealing with this problem is due in the main to the fact that it has become chiefly the concern of medical men, and in this connection the vision and the courage and determination which Dr. Bernstein has shown in his pioneer work in dealing with the defective should receive our admiration. He has demonstrated in a practical way that many of the evils which are bound up with the life and conduct of the feebleminded should justly be considered as unnecessary and avoidable evils, and that a good deal of the career of the feebleminded depends on the manner in which society deals with the problem. We should reiterate the fact that it is erroneous to approach this situation with the view that the feebleminded person is essentially vicious or criminal or antisocial, and that frequently when he becomes so it is due to the unintelligence and sometimes direct viciousness of the more intelligent members of the community in subjecting the feebleminded to tasks which he cannot possibly perform.

In these modern institutions in our state, the feebleminded person is exposed to a continuous influence of training in habit and performance which enables him to develop to his fullest capacity and to acquire ways of adjusting himself to life which I am sure eliminate many of the difficulties that are reflected in such institutions as jails, correctional houses and those dealing with the problem of illegitimacy.

I am not altogether in accord with Dr. Brown's suggestion that the public schools offer a constructive possibility in dealing with the feebleminded. I am aware, altogether too keenly, of the shortcomings of the public school system in dealing even with the normal child, and at the present time, at any rate, there is not enough energy or means available to provide for the needs of the handicapped. Moreover, the training of the feebleminded is a twenty-

four hour job, if it is to be at all constructive. One would like to see the state program develop to a point at which every feebleminded child in the community will have a period of institutional training under supervision, and from what we have heard today, I think we should be encouraged in the belief that under Dr. Brown's guidance the entire program will eventually develop to its fullest capacity.

RESEARCH WORK AT LETCHWORTH VILLAGE. DR. HOWARD W. POTTER (by invitation).

A brief history and description of Letchworth Village with special reference to its research department was given. A summary of certain studies of mental deficiency at Letchworth Village follows.

Classification of Mental Deficiency.—A classification has been adopted based on mental level, physical make-up and personality characteristics. The need for some such classification is imperative, because defective persons differ in respects other than the mental age, and hence a pertinent clinical classification is required for purposes of diagnosis, treatment, prognosis and general discussion.

Etiology of Mental Deficiency.—Based on a study of 980 cases at Letchworth Village, the etiology was apportioned as follows: In 49 per cent., there was a history of mental deficiency in the family; in 15 per cent., of insanity or epilepsy in the family; in 9 per cent., of alcoholism, antisocial behavior or dependency in the family.

In 1 per cent., there were unfavorable prenatal conditions; in 1.5 per cent., convulsions in infancy; in 5 per cent., cranial injuries at birth or early childhood; in 5 per cent., infectious diseases involving the central nervous system; in 1.5 per cent., infectious diseases other than those directly involving the central nervous system; in 8 per cent., endocrine disturbances, and in 5 per cent., no cause was found.

Personality of the Mentally Defective.—It is clear that knowing merely the mental age of a defective person tells us little as to his possibilities. A personality study of the defective person is designed not alone to classify or label him, but to supply information that may serve as a starting point or a working basis for constructive effort. A study of the personality is essential for diagnosis, understanding the basis for social and economic mal-adaptations and a useful guide for training and social direction of the defective person.

Relation of Endocrine Dysfunction to Mental Deficiency.—A clinical survey showed that 37 per cent. of the patients in the institution had some type of endocrine imbalance. Endocrine treatment is thus far not especially encouraging; only an isolated case here and there showed any tangible mental improvement.

Comparative Study of Hereditary (Primary) and Nonhereditary (Secondary) Mental Defect.—Idiots and imbeciles predominated in the nonhereditary group, and morons in the hereditary group. There was a definite retardation of physical development in the hereditary group, with no such retardation in the nonhereditary or secondary group.

The discussion following the presentation of this summary indicated two pertinent facts: (1) The marked diversity of opinions, expressed and implied, undoubtedly showed the need for a clinical classification such as is suggested above, because each speaker, while discussing the subject, was misunderstood

by the rest of the audience, despite the fact that what he stated was applicable to a certain clinical type of moron, imbecile or idiot. (2) What is most important, the discussion plainly showed our need for intensive research into this huge problem of mental deficiency.

The responsibility for this work at Letchworth Village rests with Dr. Little. If it had not been for his determination to overcome all obstacles, there would have been no research project.

CLINICAL ASPECTS OF MENTAL DEFICIENCY. DR. I. T. BROADWIN (by invitation).

This paper is based on a study of the cases seen at the mental clinic of the Department of Public Welfare, New York City. Mentally defective persons, those with epilepsy and persons presenting mental disorders of one type or another are referred here for diagnosis, disposition to the various institutions and recommendation for further home care.

The clinic averages about 1,300 new cases a year. About one half of these are diagnosed as cases of mental defect, and 10 to 15 per cent. are cases of epilepsy. A small percentage are individually represented by psychopathic and postencephalitic conduct disorders in children. The remaining number are considered as borderline cases, dull normal and normal. Many of those who were not diagnosed as mentally defective presented distinct conduct disorder problems.

The patients are examined in the usual manner. The examining physician who obtains the history from the parent or relative pays special attention to the eliciting of such facts as morbid heredity, type of birth, infectious diseases and convulsions in early infancy. The early developmental history of the patient is a subject for detailed questioning. Many of the facts obtained can bear close scrutiny in the matter of interpretation; this especially applies to morbid heredity, difficult labor, and trauma. A psychometric test is given by the psychologist. A general physical and neurologic examination is given by the examining physician. The diagnosis is established after the physician has completed a psychiatric examination. In the latter examination, the patient's character and emotional make-up are open to investigation.

In the causation of mental deficiency, the factor of morbid heredity is usually stressed. Tredgold classifies those with mental defect or amentia into two main groups: those with primary amentia, in whom the germ-plasm is defective, due to morbid heredity, constituting about 85 to 90 per cent. of the cases; and those with secondary amentia, in whom the growth of the brain has been interfered with or arrested by disease or other adverse environment. The latter group constitutes about 10 to 15 per cent. of the cases.

On closely studying the histories obtained in the clinic, it was found that the history of morbid heredity was not so frequently obtained. It was further observed that about one half of the cases presented clinical evidences of involvement of the central nervous system. The association of central nervous system involvement and mental deficiency dated from early infancy and can undoubtedly be assigned to the same cause.

These observations would apparently permit one to conclude that morbid heredity was a less frequent factor than usually considered, and that cerebral involvement due to infectious disease and other adverse environment were more than Tredgold considers them to be.

To study further the observations mentioned, 175 were divided by an arbitrary classification into two groups. Group I consisted of eighty-seven cases presenting evidence of central nervous system involvement as evidenced

by abnormal size of skull, pyramidal and extrapyramidal signs and definite cranial nerve defects. Microcephalic patients, mongols, those with definite endocrine syndromes and those with congenital syphilis were considered as admissible to this group to make it more inclusive. Group II consisted of eighty-eight cases, presenting no evidences of central nervous system involvement. In Group I, the following clinical diagnoses were made: cerebral diplegia and infantile hemiplegia, thirty-four cases; postinfectious disease, sixteen cases; Froelich syndrome, five cases; cretinism, one case; mongolism, one case; microcephalus, four cases; hydrocephalus, five cases; congenital syphilis, three cases; post-traumatic, one case; tuberous sclerosis, one case; amaurotic family idiocy, one case; unclassified, eleven cases. In the postinfectious subgroup, the infection occurred before the age of 2 years. Morbid heredity was elicited in the histories in seventeen cases.

In Group II, morbid heredity was elicited in the histories in only fifteen cases. No special significance is placed on this finding in either group, except that it does not appear to be more frequent in one group than in the other, and that it is not found in the majority of cases. Convulsions and fever in early infancy were found in eight cases. Trauma as a factor was elicited in the histories in nine other cases. This left fifty-six cases for which no apparent cause could be assigned.

The part played by such factors as fetal encephalitis, chemical toxins and trauma at birth are still matters of investigation. A large group of cerebral diplegias as described by Collier have been considered to be due to a neuronic degeneration which is self-limited in progression and which shows selectivity in the tissues involved. It may be conjectured that a similar process may be at the basis of many of the cases of mental deficiency.

The study of the cellular structure of the cortex gives promise of much that will be helpful in determining the pathogenesis of mental deficiency. The study of the etiology of mental deficiency belongs to many departments of investigation; it appears however, that the neuropathologist will be able to throw much light on this problem.

Institutionalization seems to be the logical solution to the problem of impossible adjustment to extramural life. Suitable training at home or in an institution early in life may so equip many of those with higher grade mental defects as to permit them to hold a place in life outside of an institution.

General Discussion

Dr. L. Pierce Clark: I think it is worth while saying again that it is unfortunate that investigation of the causation of feeblemindedness has been so enormously retarded in all fields of medical pathology. It is not even on an equal footing with the work in the hospitals for the insane. It is amazing to see that most of the work recorded in the textbooks has been performed in practically two cases only. It is absurd to draw any conclusions from anatomic and histologic study of the brain in this small material. It is unfortunate that feeblemindedness has been more or less annexed to psychiatry. The psychologists annexed these cases first, then the eugenists. Up to about a decade ago, it seems as if by the acummulated interest of these men, they placed at least 85 per cent. of feeblemindedness on purely hereditary lines. Slowly and gradually, this semiclinical — pathologic — psychologic theory was undermined by the accumulation of actual data to the contrary. It is down to 50 per cent. or possibly lower. That psychiatry has no province here in determining the causation of feeblemindedness, is true. We must look

at defective persons as persons with systems of feeble development, not wholly feeble development of the brain, but of the whole organism. It is a biologic problem in its largest aspects, pertaining to the whole individual and to special phases of feeble development.

The purely clinical definition of types is valuable, but no longer gives evidence that it will solve the problem as to the causation of the disease. We have to make careful studies of the metabolism in whole series of families, using experimental laboratories, and working with animals on which we can get controls, after the manner of Davenport and Stoddard. The whole issue of the causation of feeblemindedness is practically determined before birth. Therefore the problem of prevention must rest on careful research in endocrinology, metabolism, the enzyme and hormone factors, and the germ-plasm development. In order to carry out that work, I have served on a Committee connected with Letchworth Village since its beginning, and have urged various legislative bodies to give sufficient funds to carry on similar forms of research. Dr. Little and Dr. Potter have been most successful in getting small sums for investigation into the causes of mental deficiency. If the state would give the necessary equipment for investigation, it probably would not pay for the kind of personnel that is required; $40,000 a year would be needed for the personnel alone.

In regard to endocrinology, transmissive and transformative mechanisms, as physicians we are wholly aware of the enormous advance in the study of biochemistry and physiologic biology, so that we see the whole problem passing from a purely psychologic and psychiatric one into the field of experimental biology. Unless we are prepared to back this meager beginning, which has been so excellently handled, on a broad and comprehensive basis, and secure the proper personnel, we cannot get far by simple clinical and pathologic work. The National Research Council has indicated that it will make a contribution, and I hope we can get the financial means necessary for the clinical and experimental study of this problem.

Dr. Walter Timme: The problem of feeblemindedness may perhaps be roughly divided into two groups, one of which is beyond all help from the very beginning. That is the group in which feeblemindedness depends on anatomic malformation, either through injury or through hereditary conditions. The other large group is that which is functional, not organic and not anatomic. That functional group is the one which can be helped, and that functional group depends to a large extent on disease of the parents, systemic diseases, such as tuberculosis and syphilis, but to a much greater extent, on hereditary or acquired endocrinopathies. I believe Dr. Potter stated that of his group 8 per cent. were endocrinopathic patients. That was possibly a rough estimate. I advised Dr. Potter at the time of sorting the various groups, and it was rather a hurried sort of choice. To determine whether an endocrinopathy really exists is a matter depending on deep study. The fact that the patient shows superficial anomalies in growth or hair structure or skin texture or stature discrepancies and disproportions is not sufficient to determine an endocrinopathy, for the reason that a great many persons apparently normal will develop these structural manifestations that appear to be endocrinopathies as a compensatory effort for a disturbance that was preexistent, and hence we see the results of conditions which we did not know existed. Among this group of cases is one which is of tremendous importance. We have through the work of Marine and others recognized the influence of minute quantities of iodin in the early periods of life, even in intra-uterine life. The experiments on tadpoles by Gudernatsch,

Swingle, and others determined that without iodin they never become differentiated. They remain tadpoles, and may grow to be enormous in size, but they never differentiate. We feed them iodin, and they develop into their next developmental stage. We are prone to regard as the only evidence of the lack of iodin the so-called colloid goiters, chiefly seen in women in the goiter belt. It is a gross error to consider that the only manifestation. It may be the only superficial manifestation. There are only comparatively few instances in which the evidence of the lack of iodin shows as simple goiter; but there are three or four times that number of people who have no goiter who show in their entire lives this deficiency in iodin in other tissues than in the thyroid gland. I have come to classify these cases, both in my clinic and in my own work, as belonging to a special group. This group comes from the so-called goiter belt, the lack-of-iodin belt. This is much more extensive than the records published would lead us to expect. It begins at Seattle, and goes east through the Northern States, Montana and Idaho, and is of course prominent in the Great Lakes states. It goes through central New York State, through the Finger Lakes and Glens Falls, and hence to Lake Champlain and the Berkshires. There are a few patches in West Virginia and in other isolated areas. Southern Germany, Roumania, Western Russia and the Ukraine contain such belts. When you begin to divide feebleminded patients into groups according to their birthplace or that of their parents, the number that come from these regions is astonishing as compared with those from regions that so far as we know are normal. Many cases of Froehlich's dystrophy and other pronounced endocrinopathies come from these regions. These cases are not due to a deficiency of thyroid. If you examine them by the irregular and inaccurate method of basal metabolism, you may find a slight disarrangement, a rate that may be considered normal. It is not a deficiency or an excess of thyroid. It is an inefficient quality of thyroid secretion. It is a thyroid which probably lacks more than iodin, but iodin is the only element which we have recognized as being deficient. These patients represent only one group of a large number of endocrinopathic diseases that have their beginnings there. They do not all show the same picture. What the manifestations are going to be depends on how they react with their comparatively normal organs to this lack of one or another element. That is the endocrinologicopathologic problem — not a simple matter of determining whether they are acromegalic persons or dwarfs or giants. We have to recognize the original lack which has prevented them from growing up, from differentiating, from developing and from functioning, and then we will be in a fair way not only to understand this particular kind of feeblemindedness, but also to treat it. Even in later years that lack of iodin can partially be compensated for. I believe that all our efforts should be directed toward that group of remediable feeblemindedness. All the money should be spent on approaching the type that can be helped, rather than on that which cannot. Dr. Bernstein has done as much as or more than any one in helping these people to support themselves; but in spite of the fact that he says that sterilization will work just as much harm to the race through the acquiring of syphilis and gonorrhea and the transmission of these diseases through promiscuity, our problem is nevertheless to sterilize these defective persons, but at the same time to prevent or cure the syphilis and gonorrhea. Feebleminded people should not be allowed to propagate and thereby diminish by just so much the quality of the germ-plasm of those few that are still able to conduct a republic, so-called. When Dr. Bernstein further says that the moron in his opinion is man in a state of evolution, I disagree entirely with

him. I should like to ask, if this were so, and the morons were allowed to propagate among themselves, would they not reach gradually a higher state of evolution and become normal? But they do not develop. There is a defect in their germ-plasm which can never be remedied, for if it could be, and this improvement could be passed down to their offspring, we should be confronted by the transmission of acquired characteristics — a theory which has been practically exploded.

Dr. Bernard Glueck: I have little to add to what has already been said. No one who has listened to Dr. Potter's paper can fail to appreciate the significance of his researches into the causation of feeblemindedness. But there is another phase to the enterprise of the scientific study and management of the feebleminded which not only helps to clarify the problems of this group but also serves as a guide in the habit training and particularly in reeducating children who, although of normal intelligence, manifest various difficulties of behavior and adaptation. Dr. Potter's study should contribute to the important problem of conditioning human reactions, and although his work is being carried on in connection with the feebleminded, many of his findings are equally applicable to those normally constituted. The opportunities of an institution devoted primarily to research are particularly favorable for the study of the extent to which the so-called primary characteristics are fixed, organic or constitutional, and to what extent they are modifiable. This phase of the problem of behavior has had little illumination thus far, and I am sure that Dr. Potter's work is bound to contribute to its clarification.

Dr. Timme's reference to the place of the endocrine problem in the field of feeblemindedness is, of course, of tremendous importance. We know what the possibilities are for stimulating growth and development when the endocrine handicaps to this are determined and dealt with. Over and above the modifications that can be produced through endocrine therapy, however, there is the problem of habit training in social adaptation which in the last analysis is a psychologic issue, and in respect to which up to the present time the greatest contributions have come from the side of psychanalysis.

Careful, controlled work with the feebleminded should illuminate the entire problem of the conditioned reflex and the more complex problem of the socialization of human nature, and the members of this society particularly should see to it that Dr. Potter's work receives the proper recognition and encouragement.

Dr. I. J. Sands: I should like to add a few words to the discussion of feeblemindedness as I see it in private practice. As far as the etiology is concerned, we know nothing about it. The hereditary element has been mentioned, and yet nothing has been said that might shed light on the subject. We meet the mentally defective in the finest of homes and in purebred families. We all know that some eminent people have them in their families. If you call it a weakness of the germ-plasm, why should that weakness show itself in only one member and spare the rest of the family? Disturbance in the endocrine system is no more frequently found in morons than in those of normal intelligence. It is rather rare to find endocrinologic disturbances in those having mental ages between 7 and 11 years. Dr. Timme said that feeblemindedness may be due to some thyroid disturbance. As a matter of fact, the general practitioner has been taught to recognize hypothyroidism early. Hypothyroidism plays a relatively small part in mental deficiency. There are many persons of normal intelligence who have been taking thyroid

gland extracts since infancy, and there are mental defectives who have received thyroid extract without benefit. The other gland that is usually mentioned in conjunction with this problem is the pituitary. And yet in this instance, too, we have little knowledge concerning the influence of the pituitary gland on intellectual development. We know a good deal about the influence of this gland on the somatic development of the individual, but we know little of its effect on the intellectual development. Our patients presenting Froehlich syndromes are often superior in their intellectual equipment. I feel that there are two factors that play important rôles in the development of mental deficiency. One is disease in the mother during gestation — acute infectious disease that circulates toxins in the blood, such as pneumonia, typhoid fever, and nephritis — which undoubtedly affects the brain of the child. The second is the injury done to the child's brain at the time of birth. I have closely followed the work of Dr. Wilson who has performed necropsy on every stillborn child and on every child dying under one week of age. It is surprising to find the number of small hemorhages in the brains of these patients. We have searched the literature and found it rather poor about the subject of injury to the brain caused at birth. I firmly believe in Dr. Bernstein's stand on the subject, for I have followed his work closely. We may read of the opinions of an innumerable number of physicians, but whenever we wish to know of something concretely that has been done for the feebleminded, in New York State at least, we almost always go back to Dr. Bernstein's work. I fully agree with him that the defectives who show gross neurologic disturbances fall into the idiot or imbecile divisions. These two groups offer little as a problem both for study and for management. The idiot cannot exist outside of an institution, and this holds practically true in the case of the imbecile. It is the moron who lives outside of institutions who presents the real problem. The problem of the moron has assumed undue importance in recent years because of restrictions in immigration. The unskilled labor which has until recently been furnished by the immigrants, must now be supplied by the moron group of the native born. The physician, and especially the neuropsychiatrist, must help in the solution of the sociologic problem which has arisen because of restriction in immigration. As I see it, the problem of mental deficiency is really the problem of proper training in good habits and in some sort of vocation whenever possible. I fully agree with Dr. Brown that the school is the place where this training should be given.

Dr. Charles Bernstein: Dr. Timme asked a question. He said that the inheritance of acquired characteristics has never been proved. Let me recall an example. We never had a horse that could trot as fast as a horse could run until we developed one. Whenever we urged a horse to trot faster, he would break into a run. But in time we developed a horse that could trot faster than a horse could run, which was an acquired habit far beyond the natural (Redmore: Dynamic Evolution).

Regarding training the feebleminded in schools, Miss Farrell, who has made a study of this, shows what happens to the morons trained in special public schools. Somebody asked twenty years ago what of the morons in school at that time, and what has happened to them since leaving school, and it was decided to investigate just this problem, and select a place where the best school records were available. In Cincinnati, it was found boys and girls who could not make the grade or lagged behind the grade finally left school either at or before the age of 16 and went to work. Nearly all made good in

the community through serving an apprenticeship as was possible then. Now apprenticeships are limited because of changed labor conditions, and modern child labor laws require working certificates which are not issued to those under 16 years of age, except in special conditions in limited cases. Thus there is greater need for special manual, industrial and vocational training for morons. At that time they did not have child labor laws. They were giving the morons a chance for a job.

Book Reviews

PRINCIPLES OF PSYCHOTHERAPY. By DR. PIERRE JANET, Member of the Institute, Professor of the College of France. Translated by H. M. and E. R. GUTHRIE. Pp. 322. New York: The Macmillan Company, 1924.*

In 1904, Pierre Janet delivered in Boston a series of lectures on psychotherapy, which formed the basis of a large three volume work, Les médications psychologiques, published in 1920. The present brochure is an abstract of the more essential parts of that work, with especial emphasis on "the basic principles" and principal methods of psychotherapy.

In his preface, the author states that after a summary of the evolution of the methods of mental treatment will follow "a study of the psychologic phenomena and the laws on which the most interesting methods are based," and that then he "will indicate the conditions under which such methods of treatment can be applied with chances for success." This is something of an undertaking and that the author fulfils his contract may seriously be questioned. That he details a great number of the symptoms and behavior peculiarities of neurotic persons and does it in a most fluent and interesting way is true. When he comes to consider the "laws" which govern conduct and the "principles" on which psychotherapy should be based, he enters a field which still is one of controversy, not to say heated disputation. But the author seems to be scarcely aware of this fact. These laws and principles he rather expounds than "studies;" that is to say, his attitude seems to be that his conclusions are essentially the same as facts. These he explains and illustrates in an interesting and pleasing way, apparently with no thought that after all he might be wrong or that a competent person could question his conclusions or the fundamental character of his explanations. To some extent, this is indicated by his references to the literature. In the text subsequent to the historical part, of twenty-one consecutive bibliographic references, nineteen are to his own works — one to Charcot who died in 1893 and one to Gilles de la Tourette who died two or three years later. True, he occasionally refers to Dubois, Mark Twain, Déjerine, William James and others, once even to Freud, but quite casually and with no definite bibliographic reference.

But in spite of obvious lack of perspective or sense of proportion and the rather serious handicap of not being a medical man (he is a psychologist, not a physician), Janet has given us an interesting and instructive monograph.

The historical part (eighty-nine pages) begins with the temple of Aesculapius (asclepieion) at Epidaurus, touches the Middle Ages, hurries through mesmerism and magnetism and makes a long jump to Christian science, which is considered at greater length — perhaps because these lectures were delivered in Boston, possibly because "the United States seems to offer a soil favorable to the development of these cures, more magic than scientific." Then are rapidly passed in review hypnotism, the aesthesiogenies, "liquidation" of traumatic

* A review of the French edition appeared in the ARCHIVES **12**:352 (Sept.) 1924.

memories and psychanalysis, therapies derived from religious practices, reeducation, morale in medicine, faith and works, moral direction, indirect psychologic treatment and rest treatment. The whole gives a good outline of the evolution and vicissitudes of psychotherapy. For the experienced or thoughtful reader it would be quite as valuable without the interpolated explanations. A bit of pragmatism would not hurt the author. He gives us over two pages of psychologic speculation as to why hypnotism so rapidly went out of vogue as a therapeutic agent, apparently without suspecting that it was because it did not work as well as other methods.

The section on principles (155 pages) aims to elucidate the pathology of the psychoneuroses, to explain why numerous methods of mental treatment have been born to flourish for a time and then die, and to present the human psychology on which psychotherapy depends. The whole section is interesting and instructive and presents a good survey of the subject from the author's standpoint. It is scarcely necessary to add that all psychanalysts would be profoundly dissatisfied with it. Why should they not be when Janet predicts that the fate of psychanalysis will soon be that of hypnotism, metallotherapy and other fads of mental healing?

The author frankly states that the whole subject is exceedingly complex and obscure, and that all previous explanations of psychotherapy are inadequate. The reviewer thinks that Janet's own explanations do not explain. He reproaches the psychanalysts with taking refuge in novel and obscure verbiage — no new criticism — but many of his own terms and phrases are far from lucid. Just what "high tension," "asceticism that is only one form of laziness," "low psychic tension" and "psychic liquidation" may mean, is sometimes far from clear unless one is familiar with Janet's doctrinal evolution and present convictions.

But Part II does contain an amazing amount of information as to the ways and means, the manifestations and the attitude of the "neuropath" in this life, which he cannot live like his normal neighbors. There is also much practical information as to how the neuropath or psychopath protects himself from "psychic outlay" and the use of this tendency to "economy" in therapeutic methods. In somewhat recondite phraseology, the author emphasizes the old idea of "nerve exhaustion" as an essential factor in the genesis of the psychoneuroses — an idea which seems to have been gradually falling into innocuous desuetude for the last fifteen or twenty years. He says: "The problem of psychic outlay, of the cost of activity, will in the future be one of the main problems for psychology and for psychiatry: today it is hardly suspected."

Chapter IV is on "Psychic Income" — on the various ways in which a neurotic can add to his stock of vital force. Much of this chapter shows keen insight and is of real practical value, but when the author broaches the subject of stimulation, he strays from home into the fields of physiology, pharmacology and practical medicine. His disquisition on the acquisition of new tendencies is excellent, and his remarks on increase of force are helpful because suggestive; but when he likens the stimulation of alcohol (which he seems not to know is a sedative) to that of opium, cocaine, a mental attitude, a sudden emergency requiring prompt action, eating and breathing, he is quite unconvincing and occasionally peculiarly inept.

Part III concerns "The Results of Psychotherapy" (thirty-two pages). In it Professor Janet "endeavors to find" (concerning mental treatments) "under what conditions their effectiveness may be increased and to verify whether or not there are certain cases in which they are of considerable value."

First, there is a somewhat lukewarm endorsement of hypnotism based on the author's results and (quite reasonably) denial of its bad effects. There follow eighteen pages on treatment of psychic exhaustion. In effect, this is a brief essay on "traumatic memories" maladjustment and readjustment, including "psychic analysis"—not psychanalysis. Part of this essay is devoted to "acute asthenias," which for the author constitute a quite mysterious group in which "suggestion, education, psycho-analytical investigation should be entirely avoided, for they are useless and generally dangerous." We suspect that if the author were a good psychiatrist, many cases in this group would not be so cryptic.

There are twelve pages on "Treatment of Psychic Hypotension." The author tries to make clear what he means by this, but when it comes to therapeusis, he begs the question, winding up with: "A certain number of depressed patients can be relieved, whether by methods of aesthesiogeny, when their application is possible, or by other forms of stimulation. Psychasthenic states with obsessions lasting for years can be transformed; attacks of depression can be much shortened. The coincidence of mental improvement with trials of these therapeutic methods is often very interesting. In the psychological treatment by aesthesiogeny and stimulation there is a whole therapy which is, no doubt, in its infancy, and is still difficult to apply, but which, in different cases, complements the forms of treatment by habit formation and by economy of force."

Familiarity with the psychoneuroses and their treatment is not attained by reading half a dozen text books and fifteen or twenty journal articles. An enormous amount of material must be canvassed, much of it rejected, some accepted, some correlated. The factors of relative value, personal equation and dialectics must be carefully weighed. This little book by Janet—an experienced, industrious and sincere psychologist—is a real contribution to the literature, and will contribute to the education of any one who will read it carefully and with an open mind.

The translation is creditably made, although there are a few errors of carelessness and unfortunate use of terms in a few instances; e. g., "mania" as applied to compulsive neuroses. There is an adequate index, which is rather rare in a French book.

DIE KREUZUNG DER NERVENBAHNEN UND DIE BILATERALE SYMMETRIE DES TIERISCHEN KÖRPERS. By PROF. DR. JACOBSOHN-LASK. Pp. 125. Berlin: S. Karger, 1924.

In the central nervous system, we recognize commissures and decussations, the first connecting like parts of the right and left sides, and the second connecting unlike parts. More accurate knowledge of the connections of these crossing fibers has made it clear that most of the so-called commissures are either decussations or mixtures of commissural and decussating fibers. In fact, the truly commissural fibers generally branch and so connect both like and unlike structures, and the decussating fibers frequently divide, sending a branch to each side of the midplane. The functions of strictly commissural fibers can readily be understood as mediating coordinated action of the bilaterally symmetrical organs of the body. But the reasons for the extensive decussations so characteristic of most types of nervous systems have never been satisfactorily presented, though many ambitious attempts have been made.

The first third of this book is devoted to a detailed review and criticism of the previous theories, all of which are fantastic, though bearing in some

cases the names of great masters—Wundt, Flechsig, Cajal, Spitzer, Radl. The author's own treatment is much better grounded. Instead of elaborating a theory in terms of the hightly specialized and terminal relations as seen in the human brain, as did those making the earlier speculations, he reviews the entire history of the nervous system, from its first appearance in coelenterates in relation to the bilateral symmetry of the body. He seeks the basis for these decussations in the organization and function of the animal body and of the nervous system in their earliest stages. Twenty-five pages are devoted to a review of invertebrate neurology and phylogenetic speculations. The next section includes descriptions of the decussations in the nervous systems of various invertebrates, and this is followed by a discussion of bilateral symmetry in general.

The simplest nervous system of coelenterates is a diffuse nerve net through which nervous impulses can be transmitted in any direction. From such a plastic system, the nervous systems of higher animals have been derived by concentration and partial isolation of the nervous elements. In this process some parts of the primitive connecting strands are preserved, including those which cross from one side of the body to the other. The condensation of nervous elements first takes the form of a ganglionic ring around the mouth, and later, with elongation and segmentation of the body, similar ganglionic condensations appear in each segment whose longitudinal and commissural connections represent survivals of the primitive diffuse nervous network. With further integration of the bodily functions, there are fusions of various segmental ganglia, a process which culminates in the massive central nervous system of vertebrates whose commissural and decussating connections have not arisen wholly de novo but have in part persisted from the beginning of the nervous system.

With the elaboration of individual members of the body special nervous centers are developed for their local control, and unified action of the body as a whole demands that these shall be connected among themselves, partly by uncrossed and partly by commissural and decussating nervous tracts. The leading segments retain their primitive dominance, and hence arise the great ascending and descending crossed and uncrossed conduction systems. These fibers primitively divide into homolateral and heterolateral branches, and this arrangement is preserved in most of the long tracts of higher vertebrates.

The author builds his conception on the ringlike arrangement of the peripheral nervous system of the segmented invertebrates, whose leading ganglia are likewise arranged as a ring around the esophagus. Accepting the views of Gaskell and others, he regards the vertebrate central nervous system as built up around a primitive digestive tube which becomes the ventricular system of brain and spinal cord. It may be added that the fundamentals of his argument as paraphrased in the preceding paragraphs may be accepted without invoking those speculative theories of vertebrate ancestry which derive this phylum from annelids, articulates or other highly specialized segmented invertebrates.

The long pathways of the vertebrate brain in general decussate partially or wholly. A review of the evidence leads to the conclusion that the partial decussation is the primitive arrangement with increasing proportions of crossed fibers in higher vertebrates. In fact, there are few complete decussations, the trochlear nerves and the optic nerves of lower vertebrates being the most noteworthy. These cases are reviewed, though without reaching any satisfying conclusions.

THE ELEMENTS OF SCIENTIFIC PSYCHOLOGY. By KNIGHT DUNLAP. St. Louis: C. V. Mosby Company, 1922.

Dr. Dunlap's work must be regarded as one of the important contributions of the day among psychologic books. It is written as a textbook to impart to students the fundamental principles of general psychology, and one of its chief aims apparently is to impress on them at the beginning the scientific point of view in psychology. The book represents an appreciable advance in the direction of clear concepts and definitions and a laudable attempt to confine the materials for the introduction to psychology to the established facts and generalizations. That the author has kept himself free from all theoretical interpretation, or that any one could write a general textbook on psychology at the present time without making some debatable assumptions cannot be claimed.

As a textbook, this work has limitations imposed by the scientific ideal that the author has set up for himself to follow. The most conspicuous result is the disproportionate attention to the various topics necessarily treated in a textbook covering the general field, and particularly the scant treatment of the instincts and emotions attracts attention — a neglect that is consistent, however, with the author's scientific standards.

In a general way, the familiar division of psychology into structural and functional appears in this textbook. The first half of the book is mainly a statement of the accepted facts in the psychology of the senses and the thought processes. The program is not quite carried through, since the affective experiences are left to the second part. The plan of organization seems to be less clear in the second part than in the first part, and the reasons for the order of treatment of topics are not apparent. Bodily mechanisms and reaction are first considered, then the instincts, then the processes involved in perception and thought and finally the affective experiences and the self. There is the same disproportionate attention here to perception that is found in the first part.

The impression made by the work as a whole, considering it as a textbook, is that it is an excellent introduction to the psychology of the senses. The writer has, however, carried out somewhat too rigorously his plan of scientific criticism of the materials of psychology, especially in the chapters on instinct and feeling. The result is a book which, however excellent in the quality of what it includes, suffers by the absence of that which is excluded to such an extent that it does not represent human nature in the comprehensive way it should in order to rouse deep interest as an introductory textbook in psychology.

There is an appendix on mental diseases and mental deficiency which does not appear to be up to the high standard set by the remainder of the book. The classification of mental diseases is misleading: Paranoia and paraphrenia, it might be inferred, are equally important and as secure in concept as dementia praecox and manic-depressive psychosis. The achievements of the psychanalytic schools are entirely omitted.

It is not the intention of this review to criticize the assumptions and points of view of the book, but only to indicate most briefly what these are. Content, awareness, and the ego are assumed as fundamentals. Two types of elements, and only two, are recognized: sentienda and relations. The affective experiences are reduced to sentienda, and pain for the most part to nonspecific sensory activity. Experience is treated consistently as integrative.